STEADY STATE AND STABILITY ANALYSIS OF RESPIRATORY CONTROL SYSTEM USING LABVIEW

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ABSTRACT

The human respiratory system is a well developed and complex system which is constantly being perturbed by physiologic disturbances. Engineering performed good support in modeling and simulation of the various functional systems of the human body. The modeling of respiratory of the human body is required to understand its operation and pathologies present. This paper describes a steady state model and cheyne stokes breathing model of respiratory control system. Both models are simulated in LabVIEW. The steady state model gives steady state values of O_2 and CO_2 at sea level and high altitudes which are useful to provide sufficient ventilation to the persons at high altitudes and also used to analyze the stability of normal persons and congestive heart failure persons at high altitudes from cheyne stokes breathing model.

KEYWORDS

Ventilation, modeling, Gas exchanger, chemoreceptor, Respiratory controller, cheyne stokes, LabVIEW

1. INTRODUCTION

Respiratory system is a highly specific physiological sub system which is the exchange of gases O_2 and CO_2 through the act of breathing. The behavior of this system is defined by the continual interaction of the controller and peripheral processes which is similar to closed loop system. The respiratory system is well defined with peripheral controlled processes and central controller, based on the empirical analysis the respiratory control system. The engineers can be design and develop mathematical models of human body that is done through simulation software which is validated by medical specialists [1]. The behavior of the respiratory when a flow of air inside called inhalation and the movement of gas determine the mechanics of ventilation. This is very helpful for diagnosis and prediction of chronic diseases. The model used in this work is analyzed the steady state condition of human at sea level and high altitude cases, with these results, we developed a proposal which are useful for control the ventilation. This level of design for intensive care ventilators and special ventilators used in chronic care units. Generally, the respiratory control system is modeled as a closed loop feedback/feed forward regulator.

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2. ANATOMY OF RESPIRATORY TRACT

The respiratory system with upper respiratory tract includes the nose, nasal cavity, ethmoidal air cells, fronal sinuses, maxillary sinus, sphenoidal sinus, larynx and trachea. The lower tract of respiratory system consists of lungs, air ways (bronchi and bronchioles) and air sacs (alveoli). In this, the right lung is divided in to three sections, called lobes and similarly the left lung is divided in to two lobes. The passage of air from atmosphere to lungs is given below.

Initially the air enters in to the body through the nose or mouth and travels down to the throat through larynx and trachea. Then it enters in the lungs through main stem bronchi which is available in both left and right lungs. In the lungs, these main stem bronchi divided in to smaller bronchi and again divided in to smaller tubes called bronchioles. These bronchioles end with tiny air sacs called alveoli. There are several millions of alveoli in each lung, and these areas which are responsible for gas exchange. Each alveolus is closely interacted with a capillary network which contains deoxygenated blood from the pulmonary artery [2].

Basically the lung diseases are obstructive and restrictive types. In obstructive lung diseases, the person may feel hard to exhale all the air in the lungs. In case of restrictive lung diseases, the person has difficulty to fully expand their lungs with air. The most general causes of obstructive lung diseases are cystic fibrosis, asthma, bronchiectasis and chronic obstructive pulmonary disease (COPD) [3]. In restrictive lung disease occurred due to several conditions like obesity, neuromuscular disease and interstitial lung disease, etc.

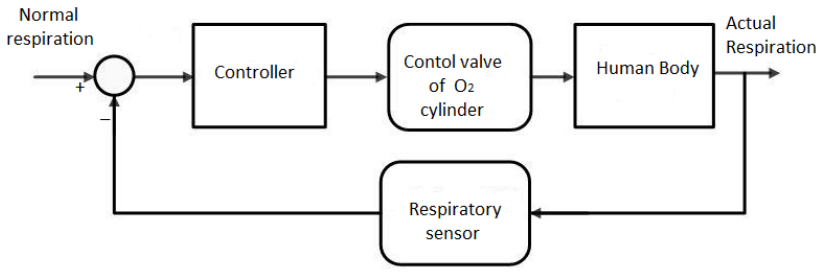

Figure 1: Closed Loop Control

So far, we have discussed the diseases related to lungs and we developed an automatic closed loop control as shown in Fig.1. This is useful in mountain trekkers and sea divers for automatic opening /closing of the oxygen cylinder valve. The oxygen is the process variable which is to be maintained based on the need of the human body. Here, the respiratory output from the human body is sensed by the respiratory sensor which is fed to the controller. Based on the error the controller facilitates the required amount of oxygen to the lungs. Initially, we developed a model for controller later; we simulated the entire model of entire closed loop respiratory closed loop systems in lab view. The simulation results are stored in the look up table and based on the error, the controller produces desired output for proper functioning of control valve. Based on the superiority of the lab view over all other simulink tools, we have chosen the lab view for this application. The modeling of the respiratory system is given clearly in the next section.

3. MODELING

The PaCO2 is measured by arterial blood gases. Partial pressure means to the pressure exerted by a specific gas ion a mixture of other gases. Then, Paco2 simply shows the measurement of CO2

in arterial blood. A rise in PaCO2 by 1mm of Hg may increase the ventilator output by a third of its resting level. However, at high altitude during inhalation of a gas mixture containing low O2 content, even normal person needs additional drive to breathe due to hypoxia. Generally if the PaO2 value is less than the 70mm of Hg then healthy person may also get hypoxia. The normal range for PaCO2 is 35 to 45 mm of Hg. If PaCO2 of person is more than 45 mm of Hg, then ha has more CO2 in his blood. This is called "hypercapnia". In normal steady state condition metabolic consumption rate of both O2 and CO2 are constant. Suppose if the ventilation is drastically increased, then it leads to increase in the partial pressure of O2 and decrease in the partial pressure of CO2 which in turn lower ventilation. Therefore, negative feedback is employed for the chemical plant. The respiratory control system used for modeling is shown in figure 2 which consists of chemical plant, mechanical plant, Respiratory control pattern generator and chemoreceptor [4][5].

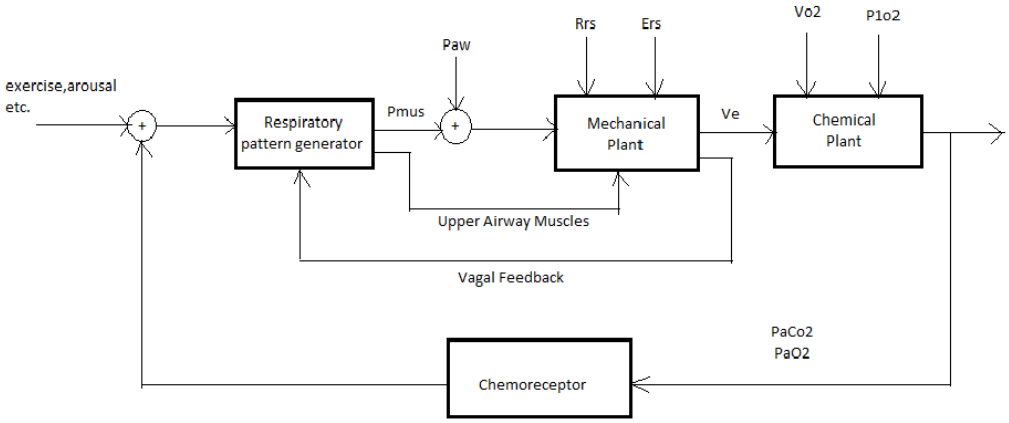

Figure 2: Respiratory control system

The input output relationships of the chemical plant are affected by the various disturbances. For example, increase in the metabolic production of CO2 due to muscular exercise. The added CO2 in the lungs is eliminated by an increase in pulmonary ventilation, the control action in restoring PaCO2 varies considerably with the type of disturbance. The sensitivity of the chemical plant depends on the load of metabolic CO2 in the lungs. The removal of CO2 from the lungs is further decreased because of perfusion mal distribution in case of pulmonary disease patients. On the other hand, metabolic acidosis and alkalosis alters arterial blood gas tensions and pH through acid- base buffering in blood. All these disturbances affect the chemical plant and leads to non linearity of the plant. The major job of the controller is to control the V_E irrespective of the disturbances with respiratory system.

Chemoreceptor consists of peripheral and central chemo receptors which are modeled by a linear first order dynamics. Peripheral receptors are having shorter response time constant and delay than central chemo receptors. The rapid changes in the blood chemistry are gated through these chemo receptors.

In mechanical plant the respiratory muscles may serve as respiratory pump. They may also act to reduce ventilation and the mechanical efficiency of respiratory muscles depends on the force-velocity and force-length relationships. The respiratory pump consists different actuators to regulate V_F in the face of many disturbances which are originating in the lungs, thorax and airways [6] [7].

The main job of respiration is to meet the metabolic demand by the exchange of O2 and CO2.The simplest controller model is a proportional controller. In the chemo reflex loop, the control signal

is a fixed relay which regulates the Ve. The stability analysis is done to a linearized system. If the loop gain and phase shift exceed unity and 180 degrees respectively then system goes to instability. The proposed model in this paper gives satisfactory responses for respiratory system. Analysis of closed loop control of ventilation and chemical plant is done through chemo reflex model in efficient manner [8].

3.1 THE MODEL FOR GAS EXCHANGER

In chemical plant, the gas exchange occurs in the lungs. From the mass balance equations of O2 and CO2, the operating characteristics of gas exchanger are obtained. The VCO2 is the metabolic CO2 production rate. The VCO2 indicates that the rate at which CO2 delivered in to the lungs is equal to the metabolic production rate. The net flow of CO2 is later equal to the difference in volumetric fractions of CO2 in the air entering and leaving the alveoli is multiplied by $V_{A.}$. Here, some portion of V_E is actually participated in the gas exchange process and some portion of total ventilation "V_D ".

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

and the CO_2 mass balance:

$$V_{CO2} = kV_A \left(F_{ACO2} - F_{ICO2} \right) \tag{2}$$

Now the constant 'k' can be is expressed as

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

The volumetric fractions F_{ACO2}, F_{ICO2} can be represented as partial pressures using Daltons law

$$P_{ICo2} = F_{ICO2} \left(P_b - 47 \right) \tag{4}$$

$$P_{ACo2} = F_{ACO2} \left(P_b - 47 \right) \tag{5}$$

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

$$P_{ACO2} = P_{ICO2} + \frac{863\mathrm{Vc}}{\mathrm{Va}} \tag{6}$$

Similarly developing mass balance equation for o_2

$$P_{AO2} = P_{Io2} - \frac{863\mathrm{Vc}}{\mathrm{Va}} \tag{7}$$

Here, P_{ACO2} - Partial pressure of alveolar O2
P_{AO2} - Partial pressure of alveolar CO2
P_{ICO2} -Partial pressure of inhaled CO2

P_{IO2} -Partial pressure of inhaled O2

Some portion of V_A is shared in both (6) and (7) equations, CO2 and O2 are independent.

3.2 THE MODEL FOR THE RESPIRATORY CONTROLLER

In the feedback section of respiratory control system shown in Fig.2 consists of chemo receptors for controlling. The generation of respiratory rhythm is dependent on the lower brain's neuronal circuits. In the gas exchange process, O2 and CO2 are independent but in case of controlling both are closely interacted. The ventilator controller output is represented as sum of the o_2 independent term and a term in which there is a multiplicative interaction between hypoxia and hypercapnia.

$$Vc = \begin{cases} \left(1.46 + \frac{32}{P_{ao2} - 38.6}\right)(P_{aco2} - 37) & P_{aco2} > 37 \\ 0 & P_{aco2} \le 37 \end{cases} \quad (8)$$

3.3 MODEL FOR CHEYNE –STOKES BREATHING

The term periodic breathing has a high importance in chronic heart failure patient's diagnosis with poor ventricular function. Periodic breathing is clusters of breaths which are separated by apnea. Cheyne stokes respiration is an abnormal breathing pattern i.e. faster breathing followed by slower breathing, in between temporary cessation in breathing.

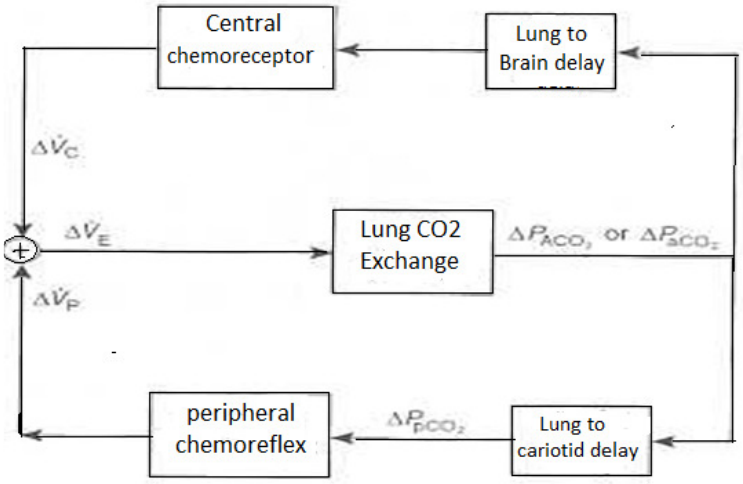

Figure 3: Respiratory control system including transportation delays

Periodic breathing does not occur commonly in normal people during wakefulness. This is characterized by periodic breathing results from instability in the feedback control system which controls the ventilation and arterial blood gases. The response of the peripheral chemo receptors is faster than the central chemo receptors. In this model we conveniently assume there are functionally two feedback loops, one from central chemo reflex and the other one from peripheral chemo reflex. First we modeled this system without transport delays and finally we added transport delays and taken the loop transfer function for stability analysis. A simplified schematic

as shown in figure.3 which consists of delays taken to transport blood from the lungs and chemo receptors [9].

MODELING OF THE LUNGS

The dynamic equivalent of the gas exchange equation is given below

$$V_L\,(dP_{ACO2}/dt)= (V_E- V_D)(P_{ICO2}- P_{ACO2})+863Q(C_{VCO2}- C_{aco2}) \quad (9)$$

Where

Q = Pulmonary blood flow
V_L = Effective CO2 storage capacity of the lungs
C_{VCO2} = Concentrations in arterial and
CaCO = Mixed venous blood

$$V_L = (d(\Delta P_{ACO2})/dt) = (V_E-V_D)\Delta P_{ACO2}+(P_{ICO2}-P_{ACO2})\Delta V_E - 863Q\Delta C_{aco2} \quad (10)$$

$$V_L\,(d\,(\Delta P_{aCO2})/dt)+(V_E-V_D+863QK_{CO2})\Delta P_{Aco2} = (PICO2- P_{aco2})\Delta V_E \quad (11)$$

Taking Laplace Transforms To Equation (5.4.3)

$$H_L\,(s) = (\Delta\, P_{aco2}/\Delta V_E) = (-G_{lL}/\,(TL.s+1)) \quad (12)$$

Where

$T_L = V_{lung}/\,(V_E-V_D+863QK_{CO2})$

$G_L = (P_{aCO2}-P_{ICO2})/\,(V_EV_D+863QK_{CO2})$

TRANSPORT DELAYS

We assume 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,

$$\Delta P_{pCO2}(t) = \Delta P_{aco2}(t-T_p) \quad (13)$$

$$\Delta P_{cCO2}(t) = \Delta P_{aco2}(t-T_c) \quad (14)$$

Applying Laplace to the Above Equations,

we get $\Delta P_{pCO2}(s) = e^{-s\tau p}\Delta P_{aco2}(s)$ (15)

$$\Delta P_{cCO2}(s) = e^{-s\tau p}\Delta P_{aco2}(s) \quad (16)$$

Dynamic response of peripheral and central chemo receptors are

$$T_p\,(dV_p/dt) +V_P = G_p\,[P_{pCO2}-I_p] \quad (17)$$

$T_c\ (dV_c/dt) + V_c = G_c\ [P_{cCO2} - I_c]$

Applying Laplace Transforms for Equations

$$\Delta V_p(s) = (G_p/(T_p s+1))\ \Delta P_{pco2}(s) \quad (18)$$

$$\Delta V_c(s) = (G_c/(T_c s+1))\ \Delta P_{c\,co2}(s) \quad (19)$$

The loop transfer functions of Lung mechanics with transportation delays are

$$H_{Lp}(s) = (\Delta V_p(s)/\Delta V_E(s)) = (G_{lung} G_p .\ e^{-s\,Tp})/(T_L s+1)(T_p s+1) \quad (20)$$

$$H_{Lc}(s) = (\Delta V_c(s)/\Delta V_E(s)) = (G_{lung} G_p .e^{-s\,Tp})/(T_L s+1)(T_c s+1) \quad (21)$$

Overall Frequency Response of the Loop Transfer Function is

$$H_L(\omega) = (\Delta V_p(\omega) + \Delta V_c(\omega))/(\Delta V_E(\omega)) \quad (22)$$

$$H_L(\omega) = (G_L/(1+j\omega T_L))\ ((G_p .\ e^{-j\omega Tp}/1+j\omega T_p) + (G_c .\ e^{-j\omega Tp}/1+j\omega T_c) \quad (23)$$

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

4. SIMULATION RESULTS

4.1. STEADY STATE MODEL

The steady state model in LABVIEW is implemented and the simulation results are shown in Figures (4), (5),(6) and (7). Equation (8) becomes progressively less valid as P_{ao2} approaches the asymptotic value 38.6 as controller output becomes infinitely large. Here case (1) corresponds to the controller output at normal sea level shown in figures (4) and (5). In case (2) gives the output of a controller in high altitude (8500 ft) is shown in figures (6) and (7).

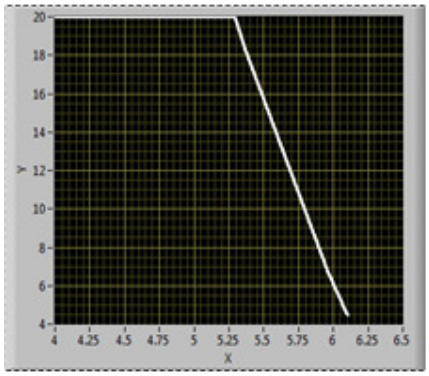

Figure 4: V_E in vs V_E out in case (1)

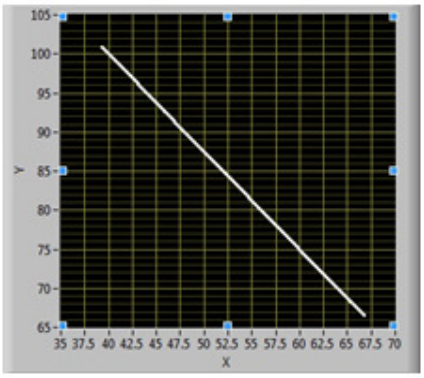

Figure 5: P_{aco2} vs P_{ao2} in case (1)

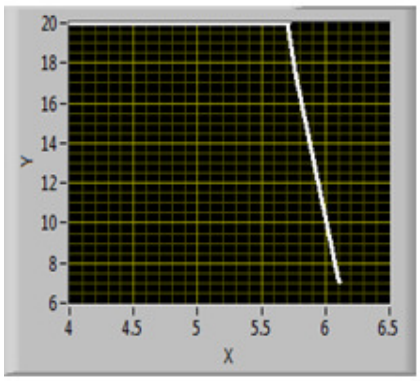

Figure 6: V_E in vs V_E out in case (2)

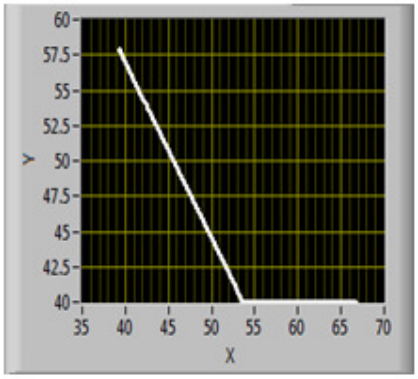

Figure 7: P_{aco2} vs P_{ao2} in case (2)

We assumed that in case (1) the person is at ground level. So the P_{IO2} value is set equal to 150mm of Hg i.e. 21% of room air, while P_{ICO2} is set equal to zero. The value of V_E affects the partial pressures of O2 and CO2. 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 below in table (1) and (2). Based on V_E value P_{ACO2} and P_{AO2} are effected i.e , if V_E increases, P_{ACO2} decreases and P_{AO2} increases.

In case (2) we consider that if the person is at high altitude (8500ft) [10]. So the P_{IO2} value is set equal to 107mm of Hg i.e.15% of room air, while P_{ICO2} is set equal to zero. Initially due low value of V_E, P_{AO2} is 40 mm of Hg and P_{ACO2} is 67mm of Hg .However due to the effect of the saturation block, P_{AO2}, is not allowed to fall below 40. 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 $P_{ACO2\ and}$ P_{AO2} for V_E case (1) and case (2) given below in tables (3) and (4).

4.2CHEYNE- 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 5) for normal and congestive heart failure persons.

Table5: parameter values used in Cheyne-stokes breathing Model.

Table1: 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

Table2: 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

Table3: 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

Table4: 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

Parameter	Value
V_L	2.5L
K_{CO2}	$0.0065 mmHg^{-1}$
G_p	$0.02 Ls^{-1} mmHg$
G_c	$0.04 Ls^{-1} mmHg^{-1}$
T_p	20sec
T_c	120sec
V_E	$0.12 Ls^{-1}$
V_D	$0.03 Ls^{-1}$
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(8) and(9).

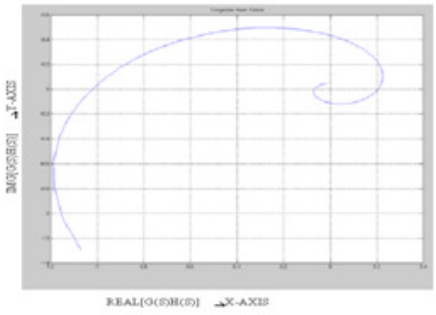

Figure 8: Normal Person

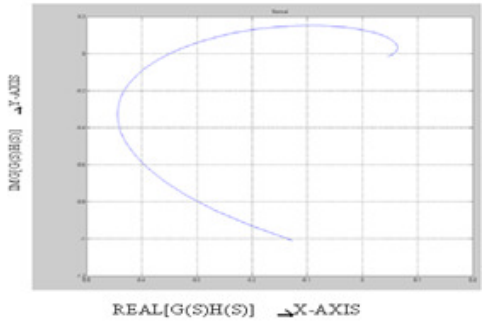

Figure 9: congestive heart failure Person

The nyquist plot shown in figures(8) and (9) represent a bandwidth of frequencies range given 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.) 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 also enters into unstable region if the frequency oscillations are more than critical frequency and the loop gain is 1.02 at critical frequency of f=0.0165 Hz and periodicity of 61 Sec These oscillations are consistent throughout the total cycle duration for normal and congestive heart failure case.

6. CONCLUSION

The steady state model demonstrates quite clearly the negative feedback nature of respiratory control. If the person is moving to high altitude levels then the oxygen content decreases and there is a need for inhale more air. These kinds of problems can be overcome by the simulated model of respiratory control system which is developed through Lab view. In this system the controller job is to calculate the steady state values of P_{ACO2} and P_{AO2}. The values are entered into look up tables and controller can be developed to control the inhalation of oxygen. By implementing this control action we can reduce the wastage of the oxygen in O_2 cylinders and we can avoid the manual control. 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.

The performance of this respiratory control system using lab view is satisfied based on the simulation results and this can also employ for some medical applications like designing an automatic ventilator for patients.

7. REFERENCES

[1] Cumming ham, D.J.C, Integrative aspects of regulation of breathing: A personal view. In: MTP International Review of Science:L Physiology series one, vol.2, Respiratory Physiology, University Park Press, Baltimore, 1974;pp. 303-369.

[2] J.Freedman H. Lehmann, R. McCormick, M. Wachter,"Respiratory Physiology". Johns Hopkins School of Medicine, USA, 1996.

[3] C.S.Poon, "Respiratory Models and Controls," in Biomedical Engineering Handbook, 2nd ed. vol. 3, Joseph D. Bronzino, Ed. CRCPress, 1995.

[4] Khoo, M.C.k., R.E.Kronauer,K.P.Strohland A.S.Slutsky. Factors inducing periodic breathing in humans: a general model.j.Appl.Physiol. 53:644-659, 1982.

[5] B.Diong,H.Nazeran,P.Nava,M.Goldman, "Modeling Human Respiratory Impedance. Comparing the Best Method with the Least Estimation Errors". IEEE Engineering In Medicine And Biology Magazine, January/February 2007
[6] Bruce EN. 1996. Temporal variations in the pattern of breathing. J Appl Physiolgy 80:1079.
[7] Balis UJ, Morris KF, Koleski J, et al. 1994. Simulations of a ventrolateral medullary neural network for respiratory rhythmogenesis inferred from spike train cross-correlation. Biol Cybern 70:311.
[8] Defares JG. 1964. Principles of feedback control and their application to the respiratory control system.In WO Fenn, H Rahn (eds), Handbook of Physiology, Respiration, sec 3, vol 1, pp 649–680,Washington, DC, American Physiological Society.
[9] NS Cherniack, JG Widdicombe (eds), Handbook of Physiology, sec 3, The Respiratory System,vol 2, Control of Breathing, part 2, pp 93–134, Washington, DC, American Physiological Society
[10] Fitzgerald RS, Lahiri S. 1986. Reflex responses to chemoreceptor stimulation. In NS Cherniack, JG Widdicombe (eds), Handbook of Physiology, sec 3, The Respiratory System, vol 2, Control of Breathing, part 1, pp 313–362, Washington, DC, American Physiological Society

Appendix

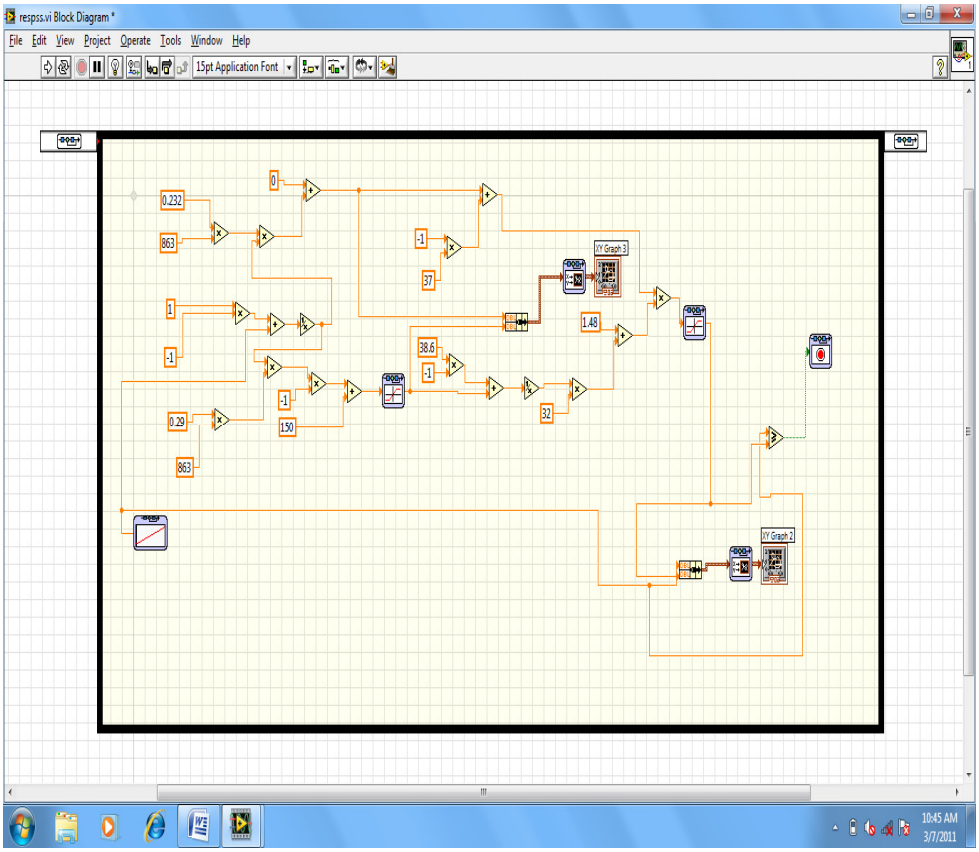

Figure4: LabVIEW Simulation for Steady state model