A mathematical model of oxygen diffusion into the blood stream

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ABSTRACT

The function of the mammalian respiratory system is the facilitation of the transfer of gas/ exchange of gas between the organism's environment and its internal transport medium, the blood. Evolutionary processes have optimized the anatomic structure of the lung as a tree-like branching network of airways terminating in thin walled elastic ducts and alveoli, where this gas exchange occurs. Here we develop a mathematical model based on the results and theories from airflow and gas exchange between the alveoli and blood capillary. The model studied the oxygen diffusion into the blood, aimed at the diffusion across the membrane. The model is based on the mass balance in the system which results in the first order linear ordinary differential equation. The model equation is solved explicitly and the, upper bound for thickness of the membrane is established.

Keywords :Oxygen ,Diffusion ,Respiratory membrane

1 INTRODUCTION

Breathing consists of two phases, inspiration and expiration. During inspiration, the diaphragm and the intercostals muscles contract. During expiration the diaphragm and muscles relax. When a breath is taken, air passes in through the nostrils, through the nasal passages, into the pharynx, through the larynx, down the trachea, into one of the main bronchi, then into smaller bronchial tubules, through the smaller bronchioles, and into a microscopic air sac called alveolus. It is here that external respiration ends.

Humans need a continuous supply of oxygen for cellular respiration, and they must get rid of excess carbon dioxide, the poisonous waste product. Gas exchange supports the cellular respiration by constantly supplying oxygen and removing carbon dioxide. The oxygen we need is derived from the Earth's atmosphere, where oxygen is 21% of the air composition. This oxygen in the air is exchanged in the body by the respiratory surface. In humans the alveoli in the lungs serve as the surface for gas exchange, into and out of the circulating blood. Air flow occurs only when there is a difference between pressures in the alveoli and the capillaries carrying the circulating blood. Air will flow from a region of high pressure to one of low pressure, where the bigger the difference, the faster the flow of the air.

Diffusion is the movement of a gas from an area of higher partial pressure concentration to an area of lower partial pressure concentration. Diffusion helps to transfer gases between (1) the lungs and the blood and (2) the blood and the peripheral tissues. The rates of diffusion of a gas across the pulmonary membranes depend on its solubility in water. For example, carbon dioxide is 21 times more soluble in water than oxygen and readily crosses the pulmonary capillary membranes [10].

In the lungs, oxygen leaves the area of higher PO_2, the alveoli, and enters the area of lower PO_2, the arterial blood in the pulmonary capillaries. Concurrently, carbon dioxide leaves the area of higher PCO_2, the

arterial blood, and enters the area of lower PCO_2, the alveoli. The blood returns via the pulmonary vein to the heart and then moves into the systemic circulation.

Gas exchange occurring in the constant-volume dead space and variable-volume collapsible and alveolar compartments was described by using species-conservation laws. On the air side of the exchanger, it was assumed that inspired air was instantaneously warmed to body temperature and fully saturated with water vapor. The gaseous mixture was presumed to obey the ideal gas law. On the blood side, it is of the same temperature with the body and fully saturated with water vapour. The gaseous mixture was presumed to obey the ideal gas law. On the blood side, the discrete constituents (plasma and erythrocytes) were lumped together and assumed to statistically behave as a uniform, homogeneous phase[1] Within a control volume, the instantaneous specific reactions were then considered to be at equilibrium; relationship between species content and their corresponding equilibrium partial pressures was consequently represented by empirical dissociation curves [3],[5] and [11]. One-dimensional axial convection provided the sole means for bulk transport of blood and movement of oxygen along the pulmonary circulation; diffusion in the radial and axial directions were ignored. Two-phase flow created due to blood heterogeneity was further disregarded. Transport of gaseous species across the alveolar-capillary membrane, assumed to be solely by diffusion, was characterized by a lumped species ling diffusing capacity , which accounted for the total diffusion-resistive path taken by species I (i=O_2, CO_2, N_2) as it diffused across the alveolar-capillary barrier, O_2 was taken up by the blood and CO_2 was excreted, whereas N_2 (a relatively inert gas) diffused in either direction, depending on the instantaneous overall ventilation-perfusion ratio [9]. The contribution of the physiological shunt [7] was neglected. The model used here was directly adapted from [4]), with the provision that alveolar pressure was not held atmospheric but rather was calculated via the airway mechanics model.

In this present study we are concerned with mathematical model of gas exchange between the alveoli and blood capillary for normal human body.

2. FORMULATION OF MODEL EQUATION

2.1. Assumptions of the model

The factors that determine the rate of gas diffusion through the respiratory membrane include;

1. The thickness of the membrane
2. The surface area of the membrane
3. The diffusion coefficient
4. The partial pressure gradient of the gas across membrane
5. The cardiac output (Blood)
6. Airflow into the lungs is periodic and is continuous
7. The respiratory membrane is cylindrical in shape

2.2. Variables and parameters of the model

P_A -alveolar gas partial pressure (mm Hg)

P_v -venous blood gas partial pressure (mm Hg)

P -partial pressure of gas at any point along the pulmonary "capillary" (mm Hg)

P_B -barometric pressure (mm Hg)

$(\Delta P)_{av}$ -average partial pressure difference between gas in alveolus and in pulmonary capillary blood (mm Hg)

C -concentration of blood gas at any point along the pulmonary "capillary" (dimensionless)

$\dot{Q}$ -cardiac output (liters/min)

ϕ -pulmonary shunt flow (liters/min)

Γ -fraction of Q which is Q8; i.e., O./Q (dimensionless)

α -solubility coefficient (atm-1)

$\frac{dQ_{O2}}{dt}$ -net rate of diffusion of oxygen across the pulmonary membrane (liters/min)

$\frac{dq}{dt}$ -net rate of diffusion of a gas into or out of the small cylinder shaped element of Fig. 2 (liters/min)

f -blood flow through a single pulmonary "capillary" (liters/min)

r -radius of a pulmonary "capillary" (meters)

χ -length of a pulmonary "capillary" (meters)

x -distance from venous blood end of "capillary" to any point along the "capillary" (meters)

n -number of equivalent pulmonary "capillaries" (dimensionless)

h -effective pulmonary membrane thickness (meters)

A -A total pulmonary capillary surface area available for diffusion (meters2)

K -constant which depends upon the physical structure of the pulmonary membrane (kg112-liters-atm/min-meters)

M - Molecular weight (kilograms)

$\dot{V}$ -oxygen consumption (liters/min)

D_C -diffusing capacity of the thin cylinder (liters/min/mmHg)

D -total diffusing capacity (liters/min/mm Hg)

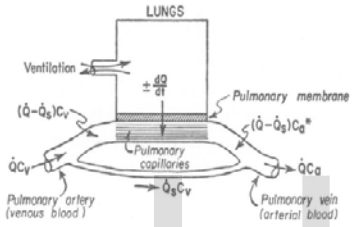
β -arbitrary constant which varies from zero at the venous blood end of pulmonary capillary to unity at the arterial blood end (dimensionless)

$2\pi r dx$ - is the surface area of the cylinder

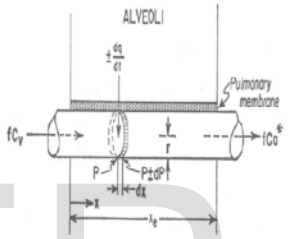
α / P_B - is the solubility coefficient expressed in $mmHg^{-1}$

2.3. The flow diagram

Fig 1.Shows a schematic diagram of the pulmonary system. Blood enters the pulmonary system at a rate Q and carries a gas of concentration C. The blood flow then splits, the major portion passing through the pulmonary capillaries where it exchanges gases with the alveoli. A small part of the blood flow, however, bypasses the capillaries and returns to the arterial blood, thereby causing the venous admixture effect.



(a) The Pulmonary System



(b) Single Pulmonary Capillary

Fig.1b above shows a schematic diagram of one of the hypothetical pulmonary capillaries. The net rate of flow of a gas across the surface area of the thin cylinder can be expressed as;

$$\frac{dq}{dt} = D_c(P_A - P) \quad \ldots\ldots(1)$$

where ΔP has been ignored, since $\Delta P << P$. The diffusing capacity of the cylinder can be expressed as a combination of factors as follows:

$$D_c = K\left(\frac{2\pi r dx}{h}\right)\left(\frac{\alpha / P_B}{M^{1/2}}\right) \quad \ldots\ldots(2)$$

Combining equations (1) and (2), we obtain

$$\frac{dq}{dt} = K\left(\frac{2\pi r dx}{h}\right)\left(\frac{\alpha / P_B}{M^{1/2}}\right).(P_A - P) \quad \ldots\ldots(3)$$

According to the law of mass balance (Fick's principle), the net rate of flow of gas across the cylinder must be equal to the blood flow f , times the blood gas concentration difference dC between the ends of the cylinder. Hence

$$\frac{dq}{dt} = fdC \quad \ldots\ldots(4)$$

Setting the right hand side of equation (3) equal to the right hand side of equation (4) rearranging, and integrating both sides yields

$$\int_{Cv}^{c}\left(\frac{1}{P_A - P}\right)dC = K\frac{2\pi\,\alpha/P_B}{fhM^{1/2}}\int_0^X dx = K\frac{2\pi r\,\alpha/P_B}{fhM^{1/2}}\,x \quad \ldots\ldots(5)$$

Examining equation 5 , we see that it contains the hypothetical quantities r , x and f . We must now eliminate these. This can be done as follows: The total number of

Parallel "capillaries" n, can be expressed as

$$n = \left(\dot{Q} - \phi/f\right) \quad \ldots\ldots(6)$$

The total capillary surface area A available for diffusion is

$$A = (2\pi r\chi)n \quad \ldots\ldots(7)$$

Combining equations 6 and 7 , eliminating n and solving for χ yields

$$\chi = \frac{Af}{2\pi r(Q - \phi)} \quad \ldots\ldots(8)$$

If we define an arbitrary constant β , such that $0 \le \beta \le 1$, we can write the distance from the venous blood end x along the capillary path as

$$x = \beta\chi \quad \text{.....(9)}$$

Where β is zero at the venous blood end and unity at the arterial blood end. Combining equations 8 and 9 yields

$$x = \beta \frac{Af}{2\pi r(Q-\phi)} = \beta \frac{Af}{2\pi r Q(1-\Gamma)} \quad \text{.....(10)}$$

in which $\Gamma = Q/\phi$

Substituting equation (10) into equation 5 yields the general equation of pulmonary Capillary gas exchange:

$$\int_{Cv}^{c} \left(\frac{1}{P_A - P} \right) dC = \beta \left(K \frac{A}{h} \right) \frac{\alpha / P_B}{M^{1/2} Q(1-\Gamma)}, \quad \text{.....(11)}$$

Where KA/h is composed of three membrane factors, K, A and h. It should be noted that the unknown hypothetical quantities r, x, f have successfully been eliminated.

For a particular gas, all constants in the right hand side of equation 11 have values which are well substantiated, except for the membrane constant KA/h. The value of this factor must, therefore, be determined. This is done as follows: The total net rate of diffusion of oxygen $\frac{dQ_{O2}}{dt}$ across the entire pulmonary membrane can be expressed as

$$\frac{dQ_{O2}}{dt} = K \frac{A}{h} \frac{\alpha / P_B}{(M_{O2})^{1/2}} (\Delta P_{O2})_{av}, \quad \text{.....(12)}$$

In which $(\Delta P_{O2})_{av}$ is the average pressure difference between alveolus and pulmonary capillary blood.

Solving equation 12 for KA/h yields

$$KA/h = \frac{(dQ_{O2}/dt)(M_{O2})^{1/2}}{\alpha_{O2}/P_B(\Delta P_{O2})_{av}} \quad \ldots\ldots(13)$$

In the steady state, $\frac{dQ_{O2}}{dt}$ is equal to oxygen consumption $\dot{V}_{O2}$. and $(\Delta P_{O2})_{av}$ can be expressed as $\dot{V}_{O2}/D_{O2}$ where D_{O2} is the diffusing capacity of oxygen. Substituting these relationships into equation 13 yields

$$KA/h = \left[\frac{(M_{O2})^{1/2}}{\alpha_{O2}/P_B}\right] D_{O2} \quad \ldots\ldots(14)$$

All of the terms on the right hand side of equation 14 have values which are well substantiated so that KA/h can now be determined.

Substituting for;

$$M_{O2} = 0.032kg, \alpha_{02} = 0.022atm^{-1}, P_B = 760mmHg, D_{O2} = 0.021liters/\min/mmHg \quad [2]$$

Yields a value of 129.3 for KA/h. K is therefore

$$K = 129.3h/A.$$

Values of $h = 0.5 \times 10^{-6}$ and $A = 70m^2$ yield a numerical value for K as approximately 9.2×10^{-7}. The reason for separating KA/h is that A/h can be maintained as a variable parameter in the final set of equations. Since the entire term KA/h will appear in the final equation, accuracy is not impaired in the Slightest by using approximate values of K and. h. Any error in the estimation of these values will be corrected by the determined value of K.

Using equation 14, changing the limits of the integral of equation 11 to partial pressures, and rearranging the right hand side, the general equation of pulmonary

Capillary gas exchange in terms of partial pressure becomes

$$\int_{pv}^{p}\left(\frac{1}{P_A - P}\right)dF(P) = \beta K\left(\frac{A}{h\dot{Q}}\right)\frac{\alpha/P_B}{M^{1/2}(1-\Gamma)}, \quad \ldots\ldots(15)$$

Where $F(P)$ denotes the function of partial pressure related to the concentration of the respective gas under study. And it is given as, according to Henry's law in blood; i.e.

$C = (\alpha/P_B)P$ .Hence (15) can be written as,

$$\int_{pv}^{p}\left(\frac{1}{P_A - P}\right)dP = \beta K\left(\frac{A}{h\dot{Q}}\right)\frac{1}{M^{1/2}(1-\Gamma)}, \quad \ldots\ldots(16)$$

It should be noted that for a given gas, all the factors in the right hand side of equation 16 are fixed, with the exception of the term $A/h\dot{Q}$.

This means that the capillary gradients can be altered by changes in surface area, membrane thickness, cardiac output, or any combination of the three.

3. METHOD OF SOLUTION

Equation 16 can be solved by direct integration as,

$$\log[P_A - P]_{Pv}^{P} = -\beta K\left(\frac{A}{h\dot{Q}}\right)\frac{1}{M^{1/2}(1-\Gamma)}$$

$$\log\left[\frac{P_A - P}{P_A - P_v}\right] = -\beta K\left(\frac{A}{h\dot{Q}}\right)\frac{1}{M^{1/2}(1-\Gamma)}$$

$$\left[\frac{P_A - P}{P_A - P_v}\right] = -\exp\left\{\beta K\left(\frac{A}{h\dot{Q}}\right)\frac{1}{M^{1/2}(1-\Gamma)}\right\}$$

$$P = P_A - (P_A - P\nu)\exp\left\{-\beta K\left(\frac{A}{h\dot{Q}}\right)\frac{1}{M^{1/2}(1-\Gamma)}\right\}$$

4. Discussion of the model

As blood flows along the pulmonary capillaries, it receives oxygen from the alveoli and delivers carbon dioxide to them. Thus, concentration gradients for these two gases develop along the pulmonary capillaries, increasing for oxygen and decreasing for carbon dioxide. Several factors, such as cardiac output, pulmonary membrane area and thickness of the membrane, affect gas diffusion into the blood stream and hence the partial pressure differences between alveolar gas and end capillary blood gas. Carbon dioxide, having a diffusion coefficient about 20 times that of oxygen, diffuses much more rapidly so that equilibrium is thought to be invariably reached, regardless of changes in these factors. Oxygen equilibrium, however, is thought to be limited either by blood flow or diffusion under certain conditions. In the flow-limiting situation, cardiac output is too great to allow equilibrium of alveolar and blood oxygen partial pressures. In the diffusion limiting situation, the pulmonary membrane, perhaps owing to a disease state such as emphysema or pulmonary congestion, is pathologically altered so that diffusion occurs too slowly to allow equilibrium to be reached between alveolar gas and blood gas. Both cases (that is, flow and diffusion limitation) may of course occur simultaneously. That is from equation 16 partial pressure related to the concentration of the respective gas under study. It should be noted that for a given gas, all the factors in the right hand side of equation 16 are fixed, with the exception of the term $A/h\dot{Q}$.

This means that the capillary gradients can be altered by changes in surface area, membrane thickness, cardiac output, or any combination. The graphs of pressure against surface area shows that the pressure gradient is increasing as the surface area is increasing until the pressure gradient reaches maximum such that increase in surface area cannot cause any further change to the pressure gradient as shown in the figure 2 below.

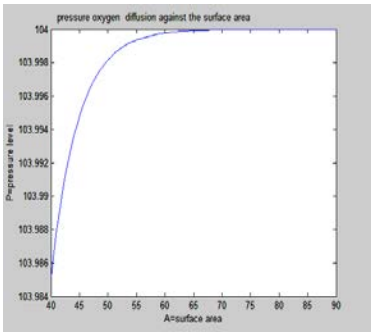


Figure 2: Effect of surface Area on gas diffusion into the blood stream

Figure 3 and 4 below shows that without changing the value of the pressure gradient, the diffusion rate through the respiratory membrane is decreasing significantly as the thickness of the membrane and Cardiac output respectively increases.

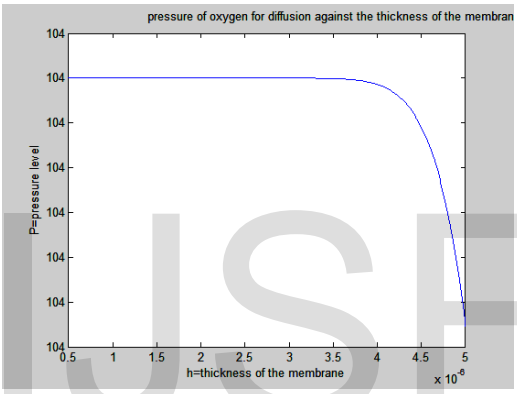


Figure 2: Effect of membrane thickness on gas diffusion into the blood stream

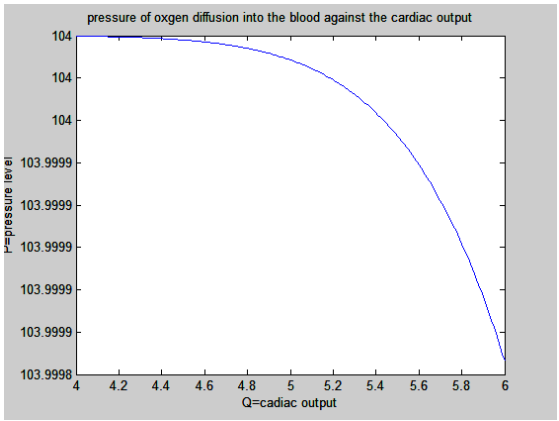


Figure 5.3: Effect of cardiac output on gas diffusion into the blood stream

5. Conclusion

From our result, increasing the thickness of the respiratory membrane above 4×10^{-6} decreases the rate of diffusion. The increase in membrane thickness can be caused by diseases.

The pulmonary edema caused by failure of the left side of the heart is the most common cause of an increase in the thickness of the membrane thereby increasing venous pressure in the pulmonary capillaries and resulting in the accumulation of fluid on the alveoli.

In a normal human, the total surface area is approximately $70m^2$ and a small decreases in the surface area adversely affects the respiratory exchange of gases. This is caused by disease condition such as emphysema and lung cancer.

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A mathematical model on the air inflow into the Lung

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ABSTRACT

Evolutionary processes have optimized the anatomic structure of the lung as a tree-like branching network of airways terminating in thin walled elastic ducts and alveoli, where this gas exchange occurs. Both dissipative and elastic properties of the respiratory system contribute to its unique static and dynamic mechanical behaviour . Here we develop a mathematical model based on the results and theories from airflow and gas exchange between the alveoli and blood capillary. The continuity and Navier- Stokes equations in the cylindrical coordinate are used to describe the flow system. The flow is assumed to be laminar and unidirectional. Consequently the continuity equation shows that the axial velocity is independent of the axial coordinates. The model equation is solved explicitly and the result show that, the flow of air through the respiratory airway is periodic.

Keywords: Airflow, respiratory airway

1. INTRODUCTION

The air we breathe passes through the trachea which divides into two main branches, the left and right bronchi, each of which again divides into ducts, each of which one again divides into two. This process continues for20-22 generations, as shown in fig. 1.Each of the terminal duct has about 300 alveolar sacs or alveoli at the end. The diameter of each alveolar sacs is between 75 and 300 microns,($10^{-6}m$ or $10^{-3}mm$).

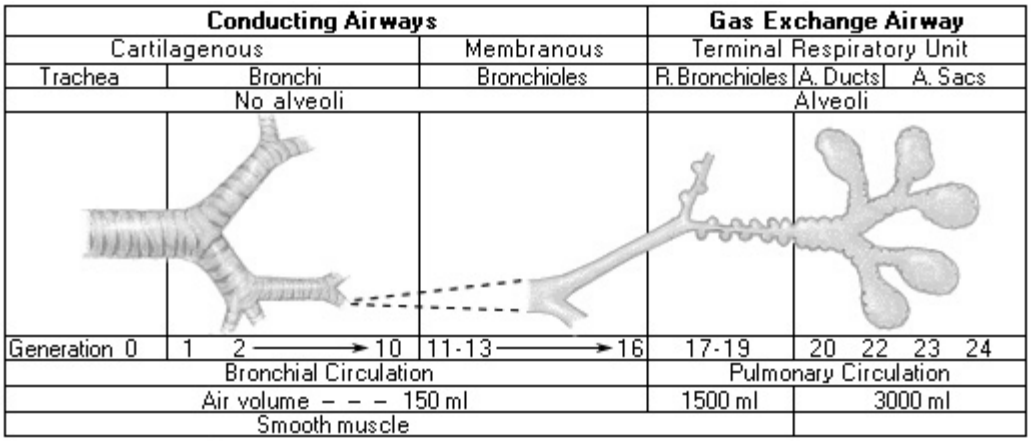

Figure 1. Airways generations Pathways

For the purposes of gas transport, the lung is divided anatomically into the conductive and respiratory zones [8] . Beginning with the trachea, the conducting airways form a series of branching conduits, including the main, lobar, and segmental bronchi, down to the terminal bronchioles. The conducting airways lead inspired air to the respiratory zone, consisting of respiratory bronchioles that contain a few budding alveoli, and finally to the alveoli-lined acini (Fig. 1). Alveoli are the terminal

gas-exchanging units of the lung. The human lung contains about 300 million alveoli, which amounts to a surface area between 50 and 100 m^2 . An acinus refers to the anatomical unit of the portion of lung distal to a terminal bronchiole.

During inspiration, a pressure gradient between the airway opening and the alveoli results in the convective movement of air from the atmosphere to the terminal units of gas exchange. For spontaneous breathing, this gradient is generated by the contraction of the diaphragm and intercostal muscles, causing the chest wall to expand and decrease the intrapleural pressure between itself and the lung. Under conditions of artificial respiration, a mechanical ventilator provides positive pressure at the airway opening relative to the alveoli. In either situation, the lung will expand and air will move convectively through the bronchial tree. While convection dominates the travel of inspired gas from the trachea throughout the conductive zone, upon reaching the respiratory zone, its velocity becomes negligible and diffusion becomes the primary means of gas transport [8] .This decrease in velocity is due to the rapid increase in total airway cross-sectional area over the short distance from the trachea to the respiratory zone. At the alveoli, inspired oxygen will diffuse down its partial pressure gradient into the bloodstream. Simultaneously, carbon dioxide will follow its diffusion gradient from the pulmonary capillaries into the alveoli to be expired.

The most important respiratory muscle is the diaphragm. The movement of diaphragm causes pressure differences and changes the volume of the lungs. During inspiration, constriction of the diaphragm muscle fibers results in a downward movement of diaphragm and the decreased pressure causes air to flow in the lungs. Normal expiration requires no muscle activation. When the diaphragm relaxes, elastic forces of the lungs and airway walls cause elastic recoil pressure and the air flows out of the lungs according to the pressure difference.

In airways the flow can be laminar, turbulent, or a combination of the two (transitional). When airflows at higher velocities especially through an airway with irregular walls, the flow is turbulent and is found mainly in the largest airways, like the trachea. When the velocity is low and goes through narrow tubes, flow is Laminar. Laminar flow is the most efficient way for oxygen transport to the distal parts of the bronchial tree. The total effective cross sectional area of the airways increases with each division of the airways from the trachea to bronchi, so that the flow rate of inspired gas falls. As the flow rate drops, the flow becomes rather Laminar than turbulent. The transition between convection and diffusion depends on the Morphometric properties of the airways so that at smallest bronchiole level (generation 15-18) the gas transport changes from convection to diffusion and gas exchanges in the alveoli [8].

[3] derives the equations for model identification in respiratory mechanics under conditions of mechanical ventilation. [7] developed physical based model of human respiration. He modeled the slow deep breathing by tunnel diode oscillator. [9] demonstrates that predictive neural network may prove to be valuable as a tool in automatic mechanical ventilation support. In his study a method suitable for an adaptive continuous control of nonlinear pulmonary mechanics has been proposed.[1]. A model of respiratory mechanics was fit to the data, and the parameters of the model are then taken to be measures of important physiological quantities. [6] developed a two-component simulation model for respiratory mechanics. A comprehensive understanding of respiratory mechanics is pivotal for the accurate diagnosis and treatment of lung disease, for adequate artificial or assisted ventilation, and for the analysis of environmental effects, like in diving or under hyperbaric conditions on lung mechanics and function.[2] stressed that gravity is important in the deposition of 0.5 and 1 micrometer particles in the human acinus.[4] incorporated gravitational sedimentation with wall movements, employing a 3-D hemispherical alveolus model. Recentl, [5] simulated trajectories of 1–5 micrometer particles in 3-D alveolated ducts representing generations 18–22 with different gravity angles. They concluded that the total deposition can be a function of the gravity angle and the ratio of the terminal settling velocity to mean lumen flow velocity. In this study we are concerned with the airflow through airways. A model integrating airflow through the air way is presented.

2. MATHEMATICAL MODEL

We model flow through the respiratory airway as a fluid defined as any material which deforms continuously under shear stress. The air is a Newtonian fluid and the dynamic viscosity μ is independent of the rate of shear. Therefore, the airflow in the respiratory system can be treated as an incompressible, viscous flow. i.e. each gas has constant volume over time, homogeneous, density of the fluid stays constant in space viscous, rate of deformation is same under the same stress for all fluid. The Velocity and the Pressure of fluid flow being a function of time and space. Airflow calculation is based on the famous Navier's-Stokes equation. We assume that the respiratory air way is cylindrical in shape. Hence we consider flow through a circular tube subject to a step change in pressure.

Therefore, the Navier- Stokes equation in the cylindrical form is given as;

$$\rho\left[\frac{\partial u_r}{\partial t}+u_r\frac{\partial u_r}{\partial r}+\frac{u_\theta}{r}\frac{\partial u_r}{\partial \theta}+u_z\frac{\partial u_r}{\partial z}-\frac{u^2\theta}{r}\right]=F_r-\frac{\partial p}{\partial r}+\mu\left[\frac{\partial^2 u_r}{\partial r^2}\frac{1}{r}\frac{\partial u_r}{\partial r}+\frac{1}{r^2}\frac{\partial^2 u_r}{\partial \theta^2}+\frac{\partial^2 u_r}{\partial z^2}-\frac{u_r}{r^2}-\frac{2}{r^2}\frac{\partial u_\theta}{\partial \theta}\right]\text{.....(1)}$$

$$\rho\left[\frac{\partial u_\theta}{\partial t}+u_r\frac{\partial u_\theta}{\partial r}+\frac{u_\theta}{r}\frac{\partial u_\theta}{\partial \theta}+u_z\frac{\partial u_\theta}{\partial z}+\frac{u_r u_\theta}{r}\right]=F_\theta-\frac{\partial p}{\partial \theta}+\mu\left[\frac{\partial^2 u_\theta}{\partial r^2}+\frac{1}{r}\frac{\partial u_\theta}{\partial r}+\frac{1}{r^2}\frac{\partial^2 u_\theta}{\partial \theta^2}+\frac{\partial^2 u_\theta}{\partial z^2}+\frac{2}{r^2}\frac{\partial u_r}{\partial \theta}-\frac{u_\theta}{r^2}\right]\text{.....(2)}$$

$$\rho\left[\frac{\partial u_z}{\partial t}+u_r\frac{\partial u_z}{\partial r}+\frac{u_\theta}{r}\frac{\partial u_z}{\partial \theta}+u_z\frac{\partial u_z}{\partial z}\right]=F_z-\frac{\partial p}{\partial z}+\mu\left[\frac{\partial^2 u_z}{\partial r^2}+\frac{1}{r}\frac{\partial u_z}{\partial r}+\frac{1}{r^2}\frac{\partial^2 u_z}{\partial \theta^2}+\frac{\partial^2 u_z}{\partial z^2}\right]\text{.....(3)}$$

With the continuity equation;

$$\frac{\partial u_r}{\partial r}+\frac{u_r}{r}+\frac{1}{r}\frac{\partial u_\theta}{\partial \theta}+\frac{\partial u_z}{\partial z}=0\text{.....(4)}$$

Since the airflow through the respiratory airway is unsteady and fluid flow is either Laminar, Navier- Stokes equation reduces to the form in axial direction in one direction only, that is $u_r=0$ and $u_\theta=0$,the continuity equation becomes,

$$\frac{\partial u_z}{\partial z}=o\text{.....(5)}$$

From equation (3) the second and the third terms on the LHS are also zeros.

The fourth term is also zero by (5). On the right hand side the third, fourth and fifth terms are zero because;

$u_z=f(r)$ Only, and there is no body force.

Hence our model now becomes:

$$\frac{\partial u_z}{\partial t}=-\frac{1}{\rho}\frac{\partial p}{\partial z}+\frac{\mu}{\rho}\left[\frac{\partial^2 u_z}{\partial r^2}+\frac{1}{r}\frac{\partial u_z}{\partial r}\right]\text{.....(6)}$$

where $\frac{\partial u_z}{\partial t}$ the rate of flow in axial direction, $\frac{\partial p}{\partial z}$ is the pressure gradient, ρ is the fluid density μ is the fluid viscosity, r is the radius of the tapered tube of the respiratory airway as in fig 2. With the boundary conditions as showed below;

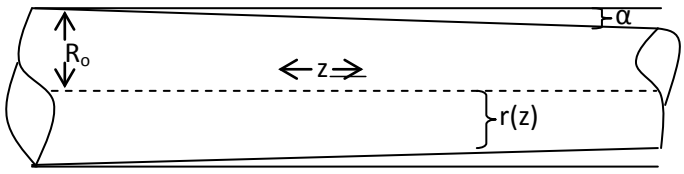

Figure 2: **Tapered tube (Trachea)**

$$r(z)=R_O-z\tan\alpha$$

$$R_O=\alpha L_I$$

$u=o$ for $r=R_O$

3. SOLUTION PROCEDURE

We shall assume that the pressure gradient causing the motion is harmonic and is given by ;

$$-\frac{1}{\rho}\frac{\partial p}{\partial z} = k\cos(t-\frac{z}{c}) \quad \text{.....(7)}$$

$$-\frac{1}{\rho}\frac{\partial p}{\partial z} = ke^{iw(t-\frac{z}{c})} \quad \text{.....(8)}$$

attributing physical significance only to the real part.

Since the motion is harmonic due to pressure gradient which is time dependent and the velocity varies with respect to z where r is tapering, velocity has the form;

$$u(r,t) = u(r)e^{iw(t-\frac{z}{c})} \quad \text{.....(9)}$$

Then to obtain the axial form of the velocity, we seek to find $u(r)$ by Substituting equation (8) and (9) into equation (6), we obtain

$$iwu(r) = k + \frac{\mu}{\rho}\left[\frac{d^2u(r)}{dr^2} + \frac{1}{r}\frac{du(r)}{dr}\right]$$

$$\frac{d^2u(r)}{dr^2} + \frac{1}{r}\frac{du(r)}{dr} - \frac{iw\rho u(r)}{\mu} = -\frac{\rho k}{\mu} \quad \text{.....(10)}$$

Equation (10) is a non-homogeneous equation which can be solved as follow;

Homogeneous part,

$$\frac{d^2u(r)}{dr^2} + \frac{1}{r}\frac{du(r)}{dr} - \frac{iw\rho}{\mu}u(r) = 0 \quad \text{.....(11)}$$

Equation (11) bears resemblance to the Bessel differential equation and thus we approximate its solution by the Bessel function of zeroeth order, with the finite solution as:

$$X = AJ_O\left(r\sqrt{\frac{-iw\rho}{\mu}}\right) \quad \text{.....(12)}$$

For the non-homogeneous par, the particular integral is given as;

$$u_p(r) = C$$

4.

such that;

$$\frac{du_p(r)}{dr} = \frac{d^2u_p(r)}{dr^2} = 0$$

Substituting these in equation (10), we have

$$\frac{-iw\rho}{\mu}C = -\frac{k\rho}{\mu}$$ and so

$$C = \frac{k}{iw} \quad \text{.....(13)}$$

Therefore, the general solution of equation (10) is;

$$u_p(r) = AJ_O\left(r\sqrt{\frac{-iw\rho}{\mu}}\right) + \frac{k}{iw}$$

Appling the boundary condition (B.C) that at

$$u = 0, r = R_o$$

We obtain

$$AJ_O\left(R_O\sqrt{\frac{-iw\rho}{\mu}}\right) + \frac{k}{iw} = 0$$

$$AJ_O\left(R_O\sqrt{\frac{-iw\rho}{\mu}}\right) = -\frac{k}{iw}$$

$$A = \frac{-k}{iwJ_O\left(R_O\sqrt{\frac{-iw\rho}{\mu}}\right)}$$

Therefore;

$$u(r) = \frac{-kJ_O\left(r\sqrt{\frac{-iw\rho}{\mu}}\right)}{iwJ_O\left(R_O\sqrt{\frac{-iw\rho}{\mu}}\right)} + \frac{k}{iw} \quad \text{.....(14)}$$

Therefore,

$$u_z = \left[\frac{-kJ_O\left(r\sqrt{\frac{-iw\rho}{\mu}}\right)}{iwJ_O\left(R_O\sqrt{\frac{-iw\rho}{\mu}}\right)} + \frac{k}{iw}\right] e^{iw(t-\frac{z}{c})}$$

$$u_z = \frac{-ik}{w} e^{iw(t-\frac{z}{c})}\left[1 - \frac{J_O\left(r\sqrt{\frac{-iw\rho}{\mu}}\right)}{J_O\left(R_O\sqrt{\frac{-iw\rho}{\mu}}\right)}\right] \quad \text{.....(15).}$$

Since $J_O(x) = 1 - \frac{x^2}{2^2} + \frac{x^4}{2^2.4^2}...$

$$\left(\left(\left(J_O\left(r\sqrt{\frac{-iw\rho}{\mu}}\right)=1-\frac{r^2\left(\frac{-iw\rho}{\mu}\right)}{2^2}+\frac{r^4\left(\frac{-iw\rho}{\mu}\right)^2}{2^2.4^2}\ \ldots\right)\right)\right)$$

Therefore considering only the first two terms we have;

$$u_z=\frac{-ik}{w}e^{iw(t-\frac{z}{c})}\left[1-\frac{\left(1+r^2\frac{iw\rho}{4\mu}\right)}{\left(1+R_O{}^2\frac{iw\rho}{4\mu}\right)}\right]..$$

$$u_z=\frac{-ik}{w}e^{iw(t-\frac{z}{c})}\left[1-\left(\frac{4\mu+r^2iw\rho}{4\mu}.\frac{4\mu}{4\mu+R^2{}_Oiw\rho}\right)\right]..$$

$$u_z=\frac{-ik}{w}e^{iw(t-\frac{z}{c})}\frac{iw\rho}{4\mu}\left[\frac{R_O{}^2-r^2}{1+\frac{R_O{}^2iw\rho}{4\mu}}\right]..$$

Or returning to the real notation,

$$u_z=\frac{k\rho}{4\mu}e^{iw(t-\frac{z}{c})}\left[R_O{}^2-r^2\right].$$

$$u_z=\frac{k\rho}{4\mu}\left[R_O{}^2-r^2\right]Cosw\left(t-\frac{z}{c}\right).....(16)$$

4. DISCUSION OF THE MODEL

During inspiration, a pressure gradient between the airway opening and the alveoli results in the convective movement of air from the atmosphere to the terminal units of gas exchange. For spontaneous breathing, this gradient is generated by the contraction of the diaphragm and intercostals muscles, causing the chest wall to expand and decrease the intrapleural pressure between itself and the lung. Under conditions of artificial respiration, a mechanical ventilator provides positive pressure at the airway opening relative to the alveoli. In either situation, the lung will expand and air will move convectively through the bronchial tree. While convection dominates the travel of inspired gas from the trachea throughout the conductive zone, upon reaching the respiratory zone, its velocity becomes negligible and diffusion becomes the primary means of gas transport. This decrease in velocity is due to the rapid increase in total airway cross-sectional area over the short distance from the trachea to the respiratory zone. At the alveoli, inspired oxygen will diffuse down its partial pressure gradient into the bloodstream. Simultaneously, carbon dioxide will follow its diffusion gradient from the pulmonary capillaries into the alveoli to be expired. During exhalation, relaxation of the diaphragm and intercostals muscles allows the elastic lung to return passively to its pre-inspiratory volume. Functional residual capacity (FRC) refers to the volume at which the inward elastic recoil of the lung is exactly balanced by the outward elastic force of the chest wall.

The many branching airway segments provide viscous pathways for airflow, and the rapid, exponential expansion in effective airway results in reduced gas velocities and changing flow regimes. The dissipative and elastic properties of the

respiratory system contribute to its unique periodic and dynamic mechanical behaviour ,that is at a fixed distance velocity time graph shows a periodic graph which represents inhalation and exhalation process in breathing mechanisms as shown in figure 6 below.

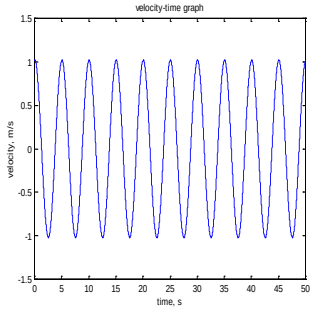

Figure 3: Periodic and dynamic flow of air through the respiratory airway

The graph below shows that, the rate of airflow into the lungs increases linearly as you go down through the respiratory airway.

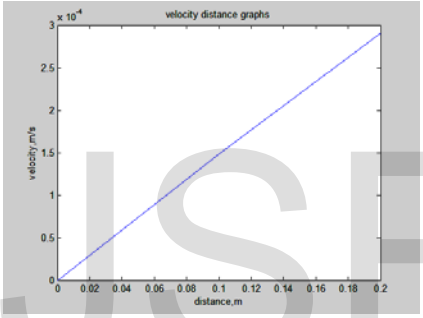

Figure 4: Periodic and dynamic flow of air through the respiratory airway

5. CONCLUSION

The flow of air through the respiratory airway is periodic at a fixed distance for one breath, in and out continuously. Thus a waveform is generated in the form of cosine function as we breath. The rate of flow is also linear at a given time.

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