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A Novel Modelling Approach to Energy Transport in a Respiratory System.

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Abstract

In this paper, energy transport in a respiratory tract is modelled using the finite element method for the first time. The upper and lower respiratory tracts are approximated as a one-dimensional domain with varying cross sectional and surface areas and the radial heat conduction in the tissue is approximated using the one dimensional cylindrical coordinate system. The governing equations are solved using one-dimensional linear finite elements with convective and evaporative boundary conditions on the wall. The results obtained for the exhalation temperature of the respiratory system have been compared with the available animal experiments. The study of a full breathing cycle indicates that evaporation is the main mode of heat transfer and convection plays almost negligible role in the energy transport. This is inline with the results obtained from animal experiments.

keywords: respiratory system, heat transfer, convection, evaporation, finite element method, stabilised method.

1 INTRODUCTION

The surface area of human respiratory tract varies between 30 and 100 m². This provides a surface to volume ratio of between 5000 and 17000 m²/m³. Such large values clearly make the human respiratory tract extremely efficient in heat and mass transport. Since mass exchange is the primary function of lungs, a large number of studies have focused on this subject. Although the energy transport is the secondary function of a respiratory tract, this is a very important function to regulate thermal balance of a body. In some animals this is one of the main method of maintaining thermal equilibrium. One of the prominent features of lungs is that approximately 700 million interconnected alveolar sacs exchange heat and gas with blood. A typical alveolus is about 200µm in size. The blood capillaries surrounding these alveolar sacs efficiently transfer carbon dioxide and heat to the air via the respiratory system. Since the temperature at the interface between blood and alveoli is approximately equal to the body temperature, the heat transfer coefficient between the alveolus and air may be calculated to be between 0.2 and 5 W/m²°C. This clearly shows that the heat transfer coefficient is lower than that of natural convection range, yet the lung is very efficient in transferring energy. Due to the highly efficient nature of heat exchange in the respiratory tracts, potential for developing compact heat exchangers by studying them in depth is high. Since engineers consider a heat exchanger with a surface to volume ratio of more than 700m²/m³ as a compact heat exchanger, the respiratory tract truly is such a heat exchanger. Some recent studies indicate that exhalation temperature of a respiratory tract may provide some indication of lung pathology. Thus, understanding the energy transport

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within a respiratory tract may be of great interest to heat exchanger researchers, biologists, ear, nose and
throat (ENT) and chest clinicians and biomedical engineers.

Many researches have attempted to measure the exhalation temperature of both humans and animals[1, 2].
It appears that the exhalation temperature may be used as a biomarker for detecting disorders of a respiratory
system. For example, in asthma patients, inflammation increases the perfusion in the airway walls and thus
more heat exchange is expected between the blood and air, resulting in a higher exhalation temperature than
normal. This hypothesis has been tested by many researchers by measuring the exhalation temperature and
 correlating it against nitric oxide (NO). The correlation is very encouraging and it appears that the exhalation
temperature of asthma patients is higher than that of normal subjects [3, 4, 5, 6, 7, 8]. These works have
demonstrated that the temperature may be used as a biomarker to determine the severity of disorders such
as asthma or chronic obstructive pulmonary disease (COPD) [9]. Such studies have been complemented
by development of temperature measurement techniques and devices [10]. Both the potential of using lung
biomimetics to develop compact heat exchangers and using exhalation temperature as a biomarker have
motivated the present modelling work. Developing a robust computational model for airway may provide
further insight into the heat transfer mechanism and allow us to manipulate important parameters. For
example, by changing the respiratory tract wall heat transfer coefficient one can easily account for increase
or decrease in heat transfer between blood and air.

A computational model of respiratory tract may be developed using three different approaches. The first
and most simple approach is empirical or lumped model approach in which correlations or zero dimensional
models are used to analyse the heat exchange[2, 11, 12, 13]. Although the lumped model is useful to
study experimental measurements and simple scenarios, it is not a robust model for predicting temperature
distribution in space and time. A more robust model may be constructed using a one-dimensional tree
with fluid-structure interaction models[14, 15] similar to blood flow analysis[16]–[28]. Such a model is very
useful when local flow distribution is of interest. The one-dimensional fluid-structure interaction has been
extensively and successfully used in systemic blood flow calculation to compute temperature[29, 30]. For
much more comprehensive and detailed study, a full three dimensional model may be employed. A fully three
dimensional model of the entire respiratory tract is extremely complex to build and thus many authors have
only focused on the upper part of the respiratory tract [31, 32, 33, 34, 35] although comprehensive lower
airway models have been recently investigate[36, 37]. While one-dimensional fluid-structure interaction
model can give a better result than the lumped models and the three-dimensional models can give every
details of the flow, both these approaches can be extremely difficult to implement to capture the details.
Since the temperature distribution in the respiratory tract is governed by a convection-diffusion equation,
an approximate way of including the velocity distribution with time and space may be sufficient to obtain
an accurate temperature distribution. Thus, a one-dimensional spatial model for temperature with varying
cross section is proposed along with a finite element solution method to solve the mathematical model.

The heat transfer mechanisms in respiratory tract include convective and latent heat losses (evaporation)[2].
The convective is a result of the temperature difference between the wall and air. An appropriate value
of heat transfer coefficient for forced convective heat transfer between the wall and air needs to be included.
The evaporative heat transfer is the result of evaporation/condensation of airway surface water. The experi-
mental work carried out on animals indicate that evaporative part is the dominant mode of heat transfer[2].
In addition to the model, including both convective and evaporative boundary conditions, a velocity wave
form needs to be prescribed along with an inlet temperature boundary condition at the inlet (note that the
inlet is also the exit). These boundary conditions should be incorporated into a convective-diffusive transport
system to determine the temperature distribution in the air. Since the respiratory tract wall temperature
will be influenced by the inhaling air temperature, assuming a fixed wall temperature along the tract will
be incorrect. Thus, a conjugate heat transfer mechanism should be included by incorporating the heat con-
duction in the surrounding soft tissue. To accurately model the heat conduction in the surrounding, the
tissue should be modelled using bioheat equations. Since the perfusion rates, metabolism and other details
in and around airway are difficult to determine, it may be possible to assume a small wall thickness with
pure heat conduction. The outer surface of the wall may be assumed to be at a temperature equal to the
body temperature. Due to the large surface area of the alveolar sacs, the temperature at the gas exchange
interface may be assumed to be equal to the body temperature. With the mentioned assumptions, a robust
model may be constructed to study the heat transport in respiratory tracts.

The paper is organised into the following sections. In the section that follows the introduction, we develop
the mathematical model in detail. In Section 3, the convection-diffusion equation is solved using a stabilized
finite element method. The results are discussed in detail in Section 4 and Section 5 derives some important
conclusions.

2 MATHEMATICAL MODEL

The two main mechanisms of heat transfer in a respiratory tract are convection and latent heat of evaporation
from the wall. The convective heat transferred from or to the respiratory tract walls may be written as [38]

$$Q_c = hP L(T_w - T_a)$$

(1)

where $h$ is the heat transfer coefficient, $P$ is the perimeter, $L$ is the length of the passage along which
heat is transferred, $T_w$ is the wall temperature and $T_a$ is the local air temperature. In a lumped fashion the
heat transfer due to convection may be written as

$$Q_c = \dot{m} c_p (T_b - T_{ai})$$

(2)

where $\dot{m}$ is the mass flow rate, $T_b$ is the body temperature and $T_{ai}$ is the inlet air temperature (ambient
temperature). Heat transfer as a result of latent heat of vaporisation may be written as [39]

$$Q_L = \lambda \dot{m} \rho_a (\phi_e - \phi_i)$$

(3)

where $\lambda$ is the latent heat of water evaporation, $\rho_a$ is the density of air and $\phi_e$ and $\phi_i$ are the absolute
humidities of expired and inspired air respectively. The absolute humidities may be defined as

$$\phi_e = \frac{10^3 M_w e^*}{R T_b}$$

(4)

and

$$\phi_i = \frac{10^3 M_w e}{R T_b}$$

(5)

where $M_w$ is the mol mass of water (18.016 g/mol), $R$ is the molar gas constant (8.3143 J/Mol K), $e^*$
(kPa) is the saturation vapour pressure of the air at $T_b$ and $e$ (kPa) is the partial vapour pressure of air at
$T_a$[40]. The vapour pressures may be calculated as [41, 2]

$$e(T) = 0.61075 \times 10^{\frac{7.5T}{T + 237.5}}$$

(6)

The total heat transferred in a respiratory tract is therefore $Q = Q_c + Q_L$[41].

Although the above lumped model is useful to study different scenarios, it is not a robust model for
predicting temperature distribution in space and time. A time dependent model in three dimensions will be
the most comprehensive model for a respiratory system. However, the structural complexities of lungs can
be extremely difficult to deal with using three dimensional spatial models. The one-dimensional models on
the other hand are simple, fast and they can provide a good overall approximation of the reality. The one-
dimensional energy equation governing temperature distribution (in degree Celsius) in a human respiratory
tract may be written for air as[38]

$$\rho_a c_p A_c \left( \frac{\partial T}{\partial t} + u \frac{\partial T}{\partial x} \right) - \frac{\partial}{\partial x} \left( k_a A_c \frac{\partial T}{\partial x} \right) + h P_a (T - T_w) + \frac{\lambda \dot{m}}{\rho_a} (\phi_b - \phi_a) = 0$$

(7)
where \( c_{pa} \) is the specific heat of air at constant pressure, \( A_c \) is the cross sectional area, \( u \) is the velocity of air, \( k_a \) is the thermal conductivity of air, \( P_a \) is the perimeter, \( T_w \) is the wall temperature, \( l \) is the length of the section in which energy is balanced, \( \phi_b \) is the local body humidity and \( \phi_a \) is the local air humidity. Since the respiratory tract wall temperature is not constant, a model to determine the wall temperature is essential. The simplest form of heat conduction model is a one-dimensional model in the radial direction originating from the respiratory tract wall. Such a model for heat conduction through the wall to determine the tissue temperature may be written as

\[
\rho_c p_c A_r \frac{\partial T}{\partial t} - \frac{\partial}{\partial x} \left( k_a A_r \frac{\partial T}{\partial x} \right) + \left[ h A_r (T - T_a) + \frac{\lambda m}{\rho_t} (\phi_a - \phi_e) \right]_{\text{interface}} = 0
\]  

(8)

where subscripts \( t \) and \( r \) indicates tissue and radial direction respectively. In the above equation, \( A_r \) is the area in the radial direction and this area is calculated using the radii along the respiratory tract and it varies depending the radial distance from the airway surface into the tissue. It should also be noted that the last two terms in Equation 8 are only used as the boundary conditions at the interface between air and tissue.

3 STABILIZED FINITE ELEMENT METHOD

Equation 7 along with appropriate initial and boundary conditions is solved in the present work using an explicit Taylor Galerkin (TG) method and the heat conduction equation 8 is solved using standard explicit Galerkin method[38, 42]. The TG method in its semi-discrete from may be written as

\[
\rho_a c_{pa} A_c \left( \frac{T^{n+1} - T^n}{\Delta t} \right) = -\rho_a c_{pa} A_c u \frac{\partial T}{\partial x}^n + \frac{\partial}{\partial x} \left( k_a A_c \frac{\partial T}{\partial x} \right)^n + \frac{u^2 \Delta t}{2} \left( \frac{\partial^2 T}{\partial x^2} \right)^n - h P_a (T - T_w)^n - \frac{\lambda m}{\rho_t} P(\phi_b - \phi_a)^n
\]  

(9)

where superscripts \( n \) and \( n + 1 \) indicates the current and next time levels between a time step \( \Delta t \). The TG effect due to convection term is retained and all other higher order terms are neglected. Assuming that the flow is incompressible and the velocity is one-dimensional and vary only as a function of cross sectional area, the temperature may be spatially discretized using linear finite elements as \( T^{(e)} = N_1 T_1 + N_2 T_2 \) in which superscript \( (e) \) indicates a linear element, \( N_1, N_2 \) are the linear shape functions and \( T_1 \) and \( T_2 \) are the nodal temperature values. The standard Galerkin weighting of Equation 9 along with the linear temperature discretization results in the following final matrix form of the convection-diffusion equation.

\[
M \left( \frac{\Delta T}{\Delta t} \right) = -T C T^n - K T^n - K_s T^n - M_c T^n + F^n
\]  

(10)

where \( M \) is the mass matrix, \( C \) is the convection matrix, \( K \) is the diffusion matrix, \( K_s \) is the stabilization matrix, \( M_c \) is the modified mass matrix for the interface convection term and \( F \) contains all the boundary conditions and evaporation term. Further details on the matrices can be found in standard finite element text books on fluid dynamics [38, 42]. In a similar fashion, Equation 8 is solved using standard Galerkin finite element method and linear elements.

3.1 Boundary conditions, solution sequence and convergence

As seen in Equations 7 and 8, the interface is linked through convective and evaporative heat transfer boundary conditions. Figure 1 (b) shows the approximate model of the respiratory tract along with the
(a) Typical respiratory tract  (b) Approximate geometry and boundary conditions

(c) Variation of cross sectional area  (d) Flow rate variation with time

Figure 1: Airway geometry details, approximation and flow rate.
boundary conditions. The convective and evaporative conditions for the temperature transport in the air (Equation 7 is applied all along the wall surface, i.e., to every finite element along the respiratory tract). However, the convective and evaporative condition for the heat conduction in the tissue, along the radial direction is only applied at the interface between the air and tissue. Also, every air node will have a solid domain in the radial direction.

As seen in Figure 1(b), for the air side, a breathing flow rate cycle is imposed at the inlet/exit, and at the interface between the air and wall, a convective heat transfer surface condition is imposed in addition to the evaporative condition. At the lung end of the tract, the temperature is assumed to be that of the body temperature at 36.8°C. As shown in Figure 1(b) every air node has a corresponding solid mesh in the radial direction. Only part of the wall of thickness of 2mm is considered in the radial direction. At 2mm from the airway surface, the tissue is assumed to reach the body temperature as shown.

The solution sequence starts with an initial temperature of the body on all tissue nodes and that of ambient air on all the air nodes. At every time step, the convection-diffusion equation, Eq.7, is solved for all air nodes and the heat conduction equation, Eq.8, is solved for all solid nodes. At the interface between the solid and air nodes, the wall temperature is used from the tissue equation to solve air temperatures and air temperature is used to solve solid wall temperatures. The convergence of the wall temperature is important to obtain a valid solution. This is achieved by carrying out the calculations for a large number of breathing cycles.

![Convergence](image)

**Figure 2:** Convergence. Respiratory air temperature distribution at the end of each breathing cycle and average exit temperature plotted against time at an ambient temperature of 50°C.

To make sure that the temperatures are converged to the right values, the calculations are carried out over a large number of breathing cycles, until the temperature change between two cycles is acceptably small as shown in Figure 2. As seen, about one hundred breathing cycles are needed to reach a converged state. Once such convergence is achieved, the uncertainties due to the initial conditions and the interface conditions are eliminated. In all the results presented in the present paper, at least two hundred breathing cycles were used.
4 RESULTS AND DISCUSSION

As shown in Figure 1, the respiratory system (Figure 1(a)) is approximated by a varying cross sectional area geometry as depicted in Figure 1(b). The varying cross sectional area is adopted from reference [43] and an approximate area variation with respiratory system length (staring from nose) is shown in Figure 1(c). The flow variation with time, during breathing, of an average human being is approximated from the measurements [44] and shown in Figure 1(d). Here, we assumed a breath rate of approximately 20 per minute representing an adult (2.9s per breath). Based the data gathered from the literature, the inlet flow variations and boundary conditions are imposed and solutions are obtained continuously for a number of breathing cycles. The converged final cycle results are used to plot the results presented below.

To make sure that the the initial transients are not influencing the final results, a convergence study in time was carried out as shown in Figure 2. The air temperatures are plotted at the end of each cycle in Figure 2. The average exit temperature over the last one third of the cycle is also plotted (referred to as 'average' in the figure). As seen, the results needed approximately one hundred breathing cycles to converge. In the remaining study we have used at least two hundred cycles to eliminate any errors associated with initial conditions. The mesh used contains very fine elements close to the inlet. Without a fine mesh close to the inlet, the calculations face an unstable solution. The parameters used in the calculations are:

- Body temperature - 36.8°C
- Thermal capacity of air, \( \rho_a C_{pa} \), - 1225 J/m\(^3\)K
- Thermal capacity of tissue, \( \rho_t C_{pt} \), - 3.8\( \times \)10\(^6\) J/m\(^3\)K
- Heat transfer coefficient between the airway wall and air, \( h \), - 0.2 W/m\(^2\)K
- Thermal conductivity of air, \( k_a \), - 0.024 W/mK
- Thermal conductivity of tissue, \( k_t \), - 0.5 W/mK
- Latent heat of water evaporation, \( \lambda \), - 2417.7 J/g
- Mol mass of water, \( M_w \), - 18.016 g/mol
- Gas constant, \( R \), - 8.3143 J/mol K

Figure 3 shows the relative importance of convection and evaporation heat transfer. In this figure the air temperature is plotted against the distance along the airway, starting from the inlet (nose). As seen the temperature distribution is dominated by evaporation heat transfer. The convection effect is very small compared to evaporation heat transfer. This very clearly confirms the experimental observation on animals[2].

Figures 4 (a) and (b) show the wall and air temperature variations over a breathing cycle at atmospheric temperatures of 20°C and -20°C respectively. The temperature values are plotted along the respiratory tract length at different times within a breathing cycle. At normal breathing conditions, the total duration of a breathing cycle assumed is 2.9s (see Figure 1(c)). The transient plots in Figure 4 shows the air and wall temperature distributions along the respiratory tract at 0.1s, 1.5s and 2.9s(0s). Up to 1.5s, the breathing cycle is in the inhalation phase and beyond 1.5s the exhalation starts. The temperature distributions shown in Figure 4 resemble a volume-pressure loop of the respiratory cycle.

Noting from Figure 4 that the temperature distributions at 2.9s is also the starting point of the breathing cycle, one can easily see the air temperature at 0.1s drops down to the ambient temperature in the vicinity of the inlet. At 1.5s (end point of inhalation), the air temperature drops down to its minimum value. At the exhalation phase, the temperature increases and reaches a maximum possible value for the given conditions. The wall temperature follows a similar pattern to that of the air temperature but with some distinctive differences. At 0.1s, the wall temperature has reduced along the length of the airway to reflect the sudden drop in inlet air temperature. This reduction in temperature is a result of inhalation air being at a lower temperature than the body temperature. This lower incoming temperature of the air starts reducing the wall
temperature from the previous cycle and the wall temperature goes down further with the air temperature at 1.5s (end of inhalation). The wall temperature then substantially increases during exhalation and reaches its maximum value at 2.9s (end of exhalation and starting of inhalation). As seen the wall temperature at the end of exhalation is higher than that of the wall temperature at 0.1s but lower than the air temperature at the end of exhalation. This is due to the extreme dynamic nature and limited residence time of air along the wall. If more air residence time (extended exhalation) is available, the wall temperature is expected to approach the air temperature. It is also obvious from both Figures 4(a) and (b) that energy transport by air movement along the respiratory tract is more efficient and faster than the heat conduction in tissue. This is another reason why wall temperature is lower than air temperature at the end of exhalation. The exhalation of air is able to rapidly transport the body temperature from the lungs to the exist (inlet).

Figure 5 also shows the results for a higher body temperature, measured on the animal model ([2] 39.8°C). Although a better match with experimental data is obtained at larger ambient temperatures, the present results deviate from experimental data at lower ambient temperatures. The largest possible uncertainty in the calculations may arise due to the imprecise input of cross sectional and surface areas and some properties. Another major unknown parameter is the heat transfer coefficient between the wall and the air. This latter uncertainty may be small as the effect of convective heat transfer on temperature is very small compared to evaporation. The assumption that the temperature of tissue reaches the body temperature at a distance of 2mm from the wall may also contribute to the uncertainties in the temperature calculations.
Figure 4: Transient temperature variation of air and wall along the respiratory tract.
5 CONCLUSIONS

In the present work, an attempt has been made for the first time to computationally model the energy transport in a respiratory tract. Although the model presented is one-dimensional in nature, the results appear to be physically meaningful and they qualitatively reflect the data produced by animal experiments[2]. The results may be further refined by including thermoregulatory mechanisms in the soft tissue. The calculations may also be improved by incorporating models for structural motion and fluid dynamics. Since the present work was aimed at only demonstrating the new model, preference was not given to study effects of various pathologies. The present model provides the basis to study effect of respiratory tract related issues such as asthma (by modifying the heat transfer coefficient between the airway wall and air) and COPD (reduction in lung surface area).

References


