ELSEVIER

Burns 32 (2006) 436–446

BURNS

www.elsevier.com/locate/burns

Theoretical evaluation of burns to the human respiratory tract due to inhalation of hot gas in the early stage of fires

Yong-Gang Lv [a,b], Jing Liu [a,*], Jun Zhang [c]

[a] *Cryogenic Laboratory, P.O. Box 2711, Technical Institute of Physics and Chemistry, Chinese Academy of Sciences, Beijing 100080, PR China*

[b] *Graduate School of the Chinese Academy of Sciences, Beijing 100039, PR China*

[c] *Laboratory for High Performance Scientific Computing and Computer Simulation, Department of Computer Science, University of Kentucky, 773 Anderson Hall, Lexington, KY 40506-0046, USA*

Accepted 2 November 2005

Abstract

A transient two-dimensional mathematical model for heat and water vapor transport across the respiratory tract of human body was established and applied to predict the thermal impact of inhaled hot gas to the nasal tissues during the early stage of fires. Influences of individual's physiological status and environment variables were comprehensively investigated through numerical calculations. Burn evaluation was performed using the classical Henriques model to predict the time for thermal injury to occur. It was shown that decreasing the air velocity and increasing the respiratory rate is helpful to minimize the burn over the respiratory tract. The effect of relative humidity of surrounding dry hot air could be ignored in predicting burns for short duration exposures. Due to evaporation cooling on the mucousal membrane, the burn often occurs at certain positions underneath the skin of the tract near the inlet of the respiratory tract. Most of the tissues near the surface suffer injury immediately after exposure to fire, while in the deeper tissues, serious damage occurs after a relatively longer time period. The method presented in this paper may suggest a valuable approach to theoretically evaluate the injury of hot air to the human respiratory tract under various fire situations.

Keywords: Burn evaluation; Fire injury; Theoretical evaluation; Respiratory tract; Bioheat transfer

1. Introduction

Burns due to inhalation of hot gas are commonly encountered, which may significantly threaten recovery of fire victims, claiming thousands of deaths in the USA alone [1]. Children, young adults, and the elderly are the most likely victims [2]. Therefore, various clinical therapies have been designed to improve survival of these patients. Strategies have also been proposed to evaluate the potential injuries of people subject to fires and thus lower the dangers. Over the past few decades, tremendous advances in burn care have contributed to a decrease in burn mortality. The new approaches include, but are not limited to, fluid resuscitation formulas [3], nutritional support regimes [4,5], high frequency oscillatory ventilation [6], introduction of localized antimicrobials, early surgical excision of eschar to reduce the occurrence of sepsis [7], and new management regimes of life-threatening hypermetabolic response to large burns [8,9]. However, no specific therapy for the combined burn and smoke inhalation injury has yet been established.

As one of the most common reasons causing death after burn, the mechanisms for the inhalation injury can be attributed to a combination of thermal, hypoxaemic and chemical effects of the hot smoke [10]. Thermal injury to the respiratory tract is usually limited to the upper respiration tract. Low heat capacity of the dry air and reflex adduction of the vocal cords protects the lower airways from thermal injury [10,11]. Clearly, a better understanding of the interplay between transient temperature and injury distribution over the human respiratory tract may help to yield a well-directed treatment in the near future. Under normal

* Corresponding author. Tel.: +86 10 62639266; fax: +86 10 62554670.
E-mail address: jliu@cl.cryo.ac.cn (J. Liu).

0305-4179/$30.00
doi:10.1016/j.burns.2005.11.006

Nomenclature

a	radius of the respiratory tract (m)
$A(z)$	airway cross section (m^2)
b	external radius of the calculation domain (m)
C	specific heat of tissue (J/kg °C)
C_A	specific heat of air (J/kg °C)
C_b	specific heat of blood (J/kg °C)
H	latent heat of water vaporization (J/kg)
h_f	heat convection coefficient between the mucousal surface and flowing air stream (W/m^2 °C)
h'_f	heat convection coefficient between the tissue and the surrounding air (W/m^2 °C)
K	thermal conductivity of tissue (W/m °C)
L	longitudinal length of human respiratory tract (m)
m	water penetrative coefficient in mucosal surface (kg/s m^2 Pa)
Nu	Nusselt number
P	pre-exponential factor (s^{-1})
$p(z)$	air perimeter (m)
p^*_a	saturated vapor pressure at surrounding air temperature (Pa)
p^*_{sk}	saturated vapor pressure at tissue temperature (Pa)
Q_m	metabolic rate of tissue (W/m^3)
R	ideal gas constant (J/mol °C)
Re	Reynolds number
r	radial position (m)
Sc	Schmidt number
t	time (s)
T	temperature (°C)
T_a	artery temperature (°C)
T_A	air temperature (°C)
T_{A0}	initial air temperature (°C)
T_0	steady-state temperature of tissue (°C)
T_f	surrounding air temperature (°C)
T_{f0}	initial surrounding air temperature (°C)
T^*	duration of a complete cycle of inspiration and expiration (s)
V	local mean volumetric longitudinal air velocity (m^3/s)
v	local mean longitudinal air velocity (m/s)
W_b	tissue blood perfusion (kg/m^3 s)
z	axial position (m)
Greek symbols	
Ω	dimensionless Henriques' burn integral
ΔE	activation energy (J/mol)
α	thermal diffusivity of tissue (m^2/s)
φ	relative humidity of surrounding air
ρ	density of tissue (kg/m^3)
ρ_A	density of air (kg/m^3)

breathing conditions, the nose and/or mouth heat and moisten the inhaled air so that its temperature is often close to the body temperature. This area usually saturates with water vapor, and recovers some of the heat and water vapor transported by the air during the expiration phase. This conditioning of the inspired air is accomplished as the airstream flow through the respiratory tract exchanging heat and water with the mucous membrane lining the airway surfaces. The process is remarkably stable over a range of inspiratory air temperature between −100 and 500 °C [12,13]. The air-conditioning process does not occur uniformly throughout the respiratory system. Most of the respiratory air-conditioning occurs in the upper respiratory tract, as shown in a previous study [14–17]. The process of thermal injury takes place quickly and the resultant injury often occurs at the early stage of fires. Accurate predictions of the early temperature elevation are, thus, very critical to evaluate burn injury in human respiratory tract under a fire. It also helps us understand what people should do to minimize lung damage when exposed to fires. Therefore, the transient air temperature history across the nose and mouth plays an important role in the thermal injury during fires. Although there have been many papers describe numerical simulation of skin burn injury, few efforts were ever made to analyze the burn process of the human upper respiratory tract. In this study, a transient theoretical model was established to describe local heat transport, and quantify the burn degree along the airway during the fires. The results might help us better understand the development of burn taking place in the respiratory tract exposed to various fire situations. To minimize lung injury when exposed to a fire or natural disaster, the time for the first-degree burns to occur is also theoretically predicted. Further, the effect of blood perfusion rate, thermal conductivity, volumetric metabolic heat of tissue, velocity and relative humidity of surrounding air and respiratory frequency was also tested.

2. Theoretical model

Analysis of heat and water transport process in the respiratory tract is generally a difficult task because the flow patterns inside this area is rather complex. For simplicity, the airway can be idealized as a long, right circular cylinder (Fig. 1). Such simplified structure had also ever been used before by other authors [14]. This is a simple however very useful treatment, which would make the theoretical analysis feasible. The tissue temperatures are considered as continuous functions of axial (z) and radial (r) positions and time (t), while the air temperatures are only the continuous functions of axial position (z) and time (t). Based on the well known Pennes bioheat transfer equation, the transient theoretical model for the present problem can be derived by applying conservation of energy to a ''slice'' across the airway, as shown schematically for the inspiration phase in Fig. 1.

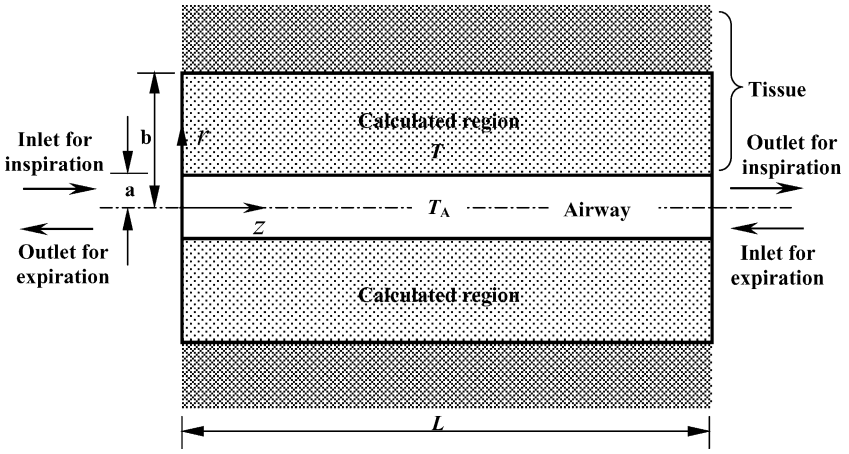

Fig. 1. Schematic model for the respiratory tract (idealized trachea).

For the tissue area, the resulting equation is accomplished as follows:

$$\frac{1}{\alpha}\frac{\partial T}{\partial t} = \frac{\partial^2 T}{\partial r^2} + \frac{1}{r}\frac{\partial T}{\partial r} + \frac{\partial^2 T}{\partial z^2} + \frac{W_b C_b}{K}(T_a - T) + \frac{Q_m}{K} \quad (1)$$

where $\alpha = K/\rho C$ is the diffusivity of tissue, ρ, C, K are, respectively, the density, specific heat and thermal conductivity of the tissue; C_b denote specific heat of blood; W_b the blood perfusion; T_a the supplying arterial blood temperature which is treated as a constant, and T the tissue temperature, and Q_m the metabolic heat generation rate.

During a real thermal process, the boundary condition (BC) at the tract skin surface is often time-dependent. At this mucous–air interface ($r = a$), the generalized boundary condition for the heat transfer is generally composed of two parts, i.e., convection and evaporation. At the entrance of the nasal cavity ($z = 0$), the continuity of the perpendicular heat flux has been imposed as a convective boundary condition. The thermoregulation mechanisms of the biological bodies have been neglected because of the slight temperature increase induced. For solving the problem, heat flux is assumed to approach zero at both the deep tissue ($r = b$) and the end of the human respiratory tract ($z = L$), which is also realistic for a biological body. Then the boundary conditions (BC) can be written as follows:

$$K\frac{\partial T}{\partial r} = h_f(T - T_A) + \frac{Hm(p^*_{sk} - \varphi p^*_a)}{1000}, \quad r = a \quad (1a)$$

$$\frac{\partial T}{\partial r} = 0, \quad r = b \quad (1b)$$

$$K\frac{\partial T}{\partial z} = h'_f(T - T_f), \quad z = 0 \quad (1c)$$

$$\frac{\partial T}{\partial z} = 0, \quad z = L \quad (1d)$$

and the initial condition (IC) is

$$T = T_0, \quad t = 0 \quad (1e)$$

where T_0 is steady-state temperature field, which is assumed as the uniform value T_a; T_f the surrounding hot air temperature; h'_f the apparent heat convection coefficient between the tissue and the surrounding air and h_f the heat convection coefficient between the mucousal surface and the flowing air stream; φ the relative humidity of surrounding air; H the latent heat of water vapour; m is water penetrative coefficient in mucosal surface; p^*_a the saturated vapor pressure at surrounding air temperature and p^*_{sk} is the saturated vapor pressure at tissue temperature.

In the respiratory tract, the air temperature gradients in the axial direction are usually much smaller than that in the radial direction. Correspondingly, the axial conduction is neglected here for simplicity. Inclusion of axial conduction to conduct a more strict analysis requires further research to possibly improve the model. Then the energy balance equation for the air can be obtained as

$$\frac{\partial T_A}{\partial t} = -v(t,z)\frac{\partial T_A}{\partial z} + \frac{p(z)}{\rho_A C_A A(z)}\left[h_f(T - T_A) + \frac{Hm(p^*_{sk} - \varphi p^*_a)}{1000}\right] \quad (2)$$

where C_A and ρ_A are, respectively, the specific heat and density of the air; $A(z)$ and $p(z)$ are the local airway cross section and perimeter, respectively; $v(t,z)$ is the local mean longitudinal air velocity ($v(t,z)$=V/A), and V the local volumetric longitudinal air velocity.

During the inspiration phase, the axial inlet boundary conditions at $z = 0$ can be considered as the boundary air temperature (T_f):

$$T_A = T_f, \quad z = 0 \quad (3a)$$

During an expiratory phase of the breathing cycle, the heat flux is assumed to approach zero at the end of the human respiratory tract ($z = L$). The boundary condition for

expiration is therefore listed as follows:

$$\frac{\partial T_A}{\partial z} = 0, \quad z = L \tag{3a'}$$

The burn process is modeled using the following initial condition:

$$T_A = T_{A0}, \quad t = 0 \tag{3b}$$

To calculate the transient air temperature field, the initial air temperature distribution T_{A0} before inhalation injury needs to be known. It can be obtained by solving Eq. (2) with the following boundary conditions:

$$T_{A0} = T_{f0}, \quad z = 0 \tag{3c}$$

in which T_{f0} is the initial surrounding air temperature.

3. Results and discussion

3.1. Solution strategy and model parameters

A predictor–corrector numerical method is applied to solve the above equations. The space and time steps are small enough to ensure that the transient temperatures are mesh-independent. Blood perfusion, thermal conductivity, and heat capacity are assumed to be constant in the tissue despite the temperature elevation. In the following calculation, the typical values for tissue and air properties are taken as [18]

$\rho = 1000\ \text{kg/m}^3$, $C = C_b = 4000\ \text{J/kg}\ ^\circ\text{C}$, $T_a = 37\ ^\circ\text{C}$, $K = 0.5\ \text{W/m}\ ^\circ\text{C}$,
$W_b = 0.5\ \text{kg/m}^3\ \text{s}$, $Q_m = 420\ \text{W/m}^3$, $h_f' = 50\ \text{W/m}^2\ ^\circ\text{C}$, $L = 60\ \text{cm}$, $T_{f0} = 20\ ^\circ\text{C}$,
$T_f = 100\ ^\circ\text{C}$, $a = 0.635\ \text{cm}$, $b = 3.635\ \text{cm}$, $V = 300\ \text{cm}^3/\text{s}$, $\varphi = 0.3$, $H = 2430.9 \times 10^3\ \text{J/kg}$,
$m = 1.27 \times 10^{-6}\ \text{kg/s m}^2\ \text{pa}$, $C_A = 1005\ \text{J/kg}\ ^\circ\text{C}$, $\rho_A = 1.165\ \text{kg/m}^3$.

The heat convection coefficient h_f between the mucosal surface and the flowing air stream is evaluated from physiological data by a naphthalene sublimation experiment [14]. The values for inspiration and expiration can be obtained from the following equations, respectively.

For inspiration phase:

$$Nu = 0.056 Re^{0.856} Sc^{1/3} \tag{4a}$$

For expiration phase:

$$Nu = 0.017 Re^{0.984} Sc^{1/3} \tag{4b}$$

When the temperature T is between 0 and 200 °C, an accurate approximation to saturated vapor pressure is [19]:

$$\ln(P^*) = \frac{C_1}{T} + C_2 + C_3 T_P + C_4 T_P^2 + C_5 T_P^3 + C_6 \ln(T_P) \tag{5}$$

where $T_P = 273.15 + T$; $C_1 = -5800.2206$; $C_2 = 1.3914993$; $C_3 = -0.04860239$; $C_4 = 0.41764768 \times 10^{-4}$; $C_5 = -0.1442093 \times 10^{-7}$; $C_6 = 6.5459673$.

In the normal respiration, the air is taken in through the nostrils without making any special effort, and sound or exaggerated movement of the nose or chest. In short, it is done unconsciously. Breathing is a cyclic phenomenon. The air temperature oscillates with alternative inspiration and expiration. The duration of a complete cycle of inspiration and expiration normally takes about 3 s, and the frequency of respiration is about 20 breaths/min, in a resting normal adult, at sea level.

3.2. Temperature responses of tissues and air in human nasal tract during a fire

Depicted in Fig. 2 is the steady-state temperature distribution of tissues along the airway in normal conditions. It can be seen that air temperature increases with distance away from the nose and at about the main bronchial generation ($z = 18$ cm), it reaches about 90% of the body temperature during the inspiration phase. The heat loss associated with the latent heat of the water vaporization may result in a local drop in temperature. For convenience, the mucosal temperature is assumed to be constant in normal condition. However, it will be seriously affected by inhaled surrounding hot air when a person is exposed to a fire or natural disaster. Figs. 3 and 4 give the transient temperature distribution in tissue and air after the exposure. Clearly, the surface tissue temperature increases immediately after the exposure, while in the deeper tissues, the temperature increases slightly until after a longer period of time. Thermal injury occurs on exposed external surface areas including the nose and mouth; burns below the trachea are nearly not encountered due to the efficiency of the upper airway in absorbing the heat. Fig. 3 still indicates that, there seems to appear no obvious temperature fluctuations throughout

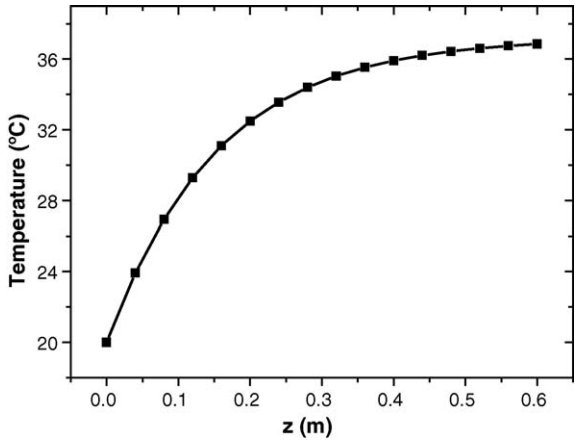

Fig. 2. Steady-state temperature distribution of tissue along the airway in normal conditions.

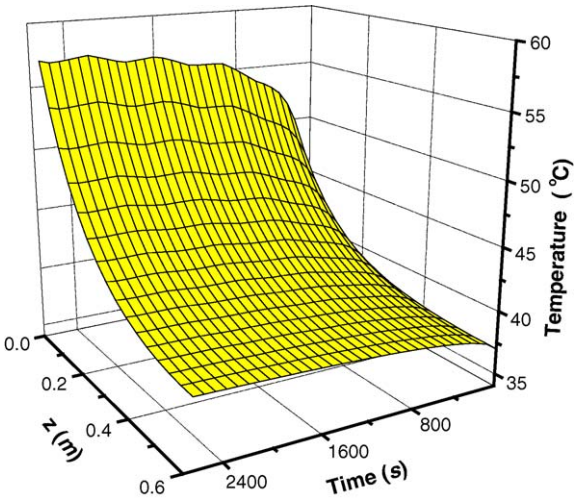

Fig. 3. Transient tissue temperature profile during a fire (K = 0.5 W/m °C, W_b = 0.5 kg/m^3 s, Q_m = 420 W/m^3, T_f = 100 °C, V = 300 cm^3/s, φ = 0.3, T^* = 3 s).

the inspiratory and expiratory phases of the breathing cycle. The reason can be attributed to the high air speed and thermal capacity of tissue. On the one hand, the air velocity during the inspiratory and expiratory phases of the breathing cycle is so high that heat exchange between air and mucosa can quickly reach its steady state. Another reason lies in that, thermal capacity of the tissue is much higher than that of the air. However, this small fluctuation can be seen clearly in Fig. 5. Due to heat loss to the tissue, the air temperature decreases acutely from the inlet of respiratory tract to the outlet during an inspiration phase. However, the air temperature remains almost the same over the expiration stage (Fig. 4). The temperature of the inspired air is decreased to that near the body core temperature (37 °C). Thus, almost no heat exchange occurs during expiration.

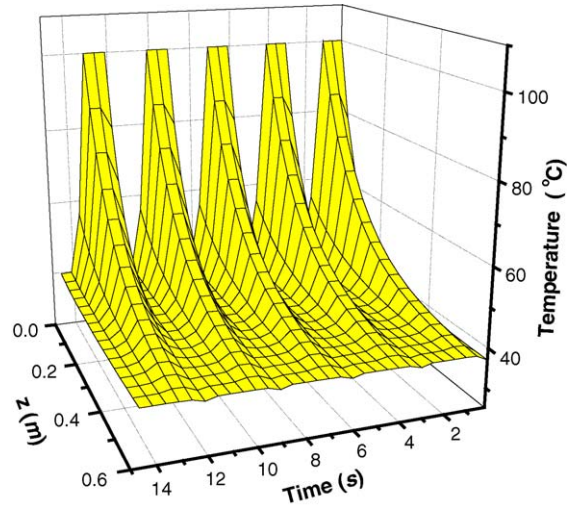

Fig. 4. Transient air temperature distribution during a fire (K = 0.5 W/m °C, W_b = 0.5 kg/m^3 s, Q_m = 420 W/m^3, T_f = 100 °C, V = 300 cm^3/s, φ = 0.3, T^* = 3 s).

In order to show the tissue temperature fluctuation clearly, transient tissue temperature responses at two specific positions z = 8 cm, r = 0.635 cm and z = 52 cm, r = 0.635 cm are particularly given in Fig. 5. One can see that the higher amplitude of the temperature oscillation occurs at the position near the inlet of the respiratory tract. Fig. 6 gives out the transient air temperature responses at the two sections z = 8 cm and z = 52 cm. The oscillations are much larger than that of the tissue temperature due to low thermal capacity of the air. In the inlet of the respiratory tract, the air temperature is mainly influenced by the surrounding air. Thus, the highest temperature almost does not change during inspiration. However, the influence of the surrounding air will become weak near the outlet of the respiratory tract where the highest and the lowest air temperatures both gradually increase with the increase of time.

3.3. Influence of individual's physiological status and environment variables

Many factors influence environmental effects on tissue temperature response. The individual's psychology and physiology are also important. Parametric analysis on these factors will help to appropriately address the burn injury issue.

Different people such as young or old have different blood perfusion rates. Even the same person has a varied blood perfusion at various health conditions. Clearly, as the main heat transfer path, the blood perfusion level will affect the biological response to a fire. Fig. 7 depicts the tissue temperature responses corresponding to three different blood perfusion levels. For the case of W_b = 5.0 kg/m^3 s, the tissue temperature appears much smaller than that of using W_b = 0.05 kg/m^3 s at the same position. It can be seen that large blood perfusion tends to prevent the biological body from burn injury. From this point, one can predict that an old person, whose perfusion is generally lower, is easier to be injured by a fire than a young person is. This is because the higher blood perfusion compensates for the heat produced by the external surrounding hot air. In the same time, one can find that the time for the tissue temperature to reach a steady state is shorter when the blood perfusion is higher. For the case of W_b = 0.5 kg/m^3 s, about 420 s is needed for the tissue temperature to reach a steady state. However, when the blood perfusion is lower (W_b = 0.05 kg/m^3 s), a much longer time is needed. The result can help explain the fact that the elderly [2] are among the most likely victims of thermal injury. Further calculation shows the influence of the thermal conductivity on the tissue temperature response (Fig. 8). The tissue temperature increases with the increase of the thermal conductivity of tissue. The temperature of the surface tissue will be closer to the body core temperature when the thermal conductivity becomes high enough. In fact, the thermal conductivity of the tissue depends on its components. The

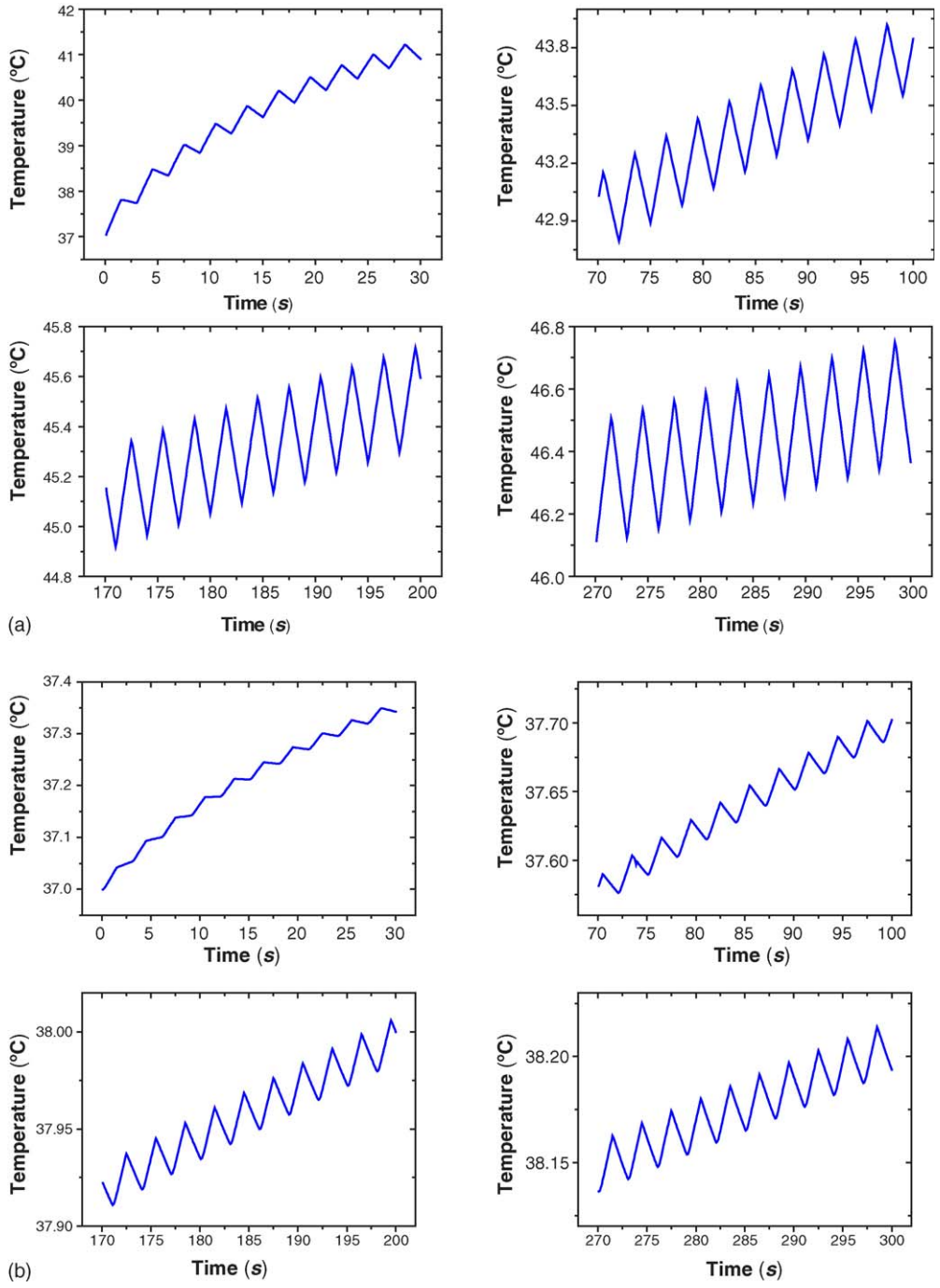

Fig. 5. Transient tissue temperature responses at two specific positions (a) $z = 8$ cm, $r = 0.635$ cm and (b) $z = 52$ cm, $r = 0.635$ cm at four time segments ($K = 0.5$ W/m °C, $W_b = 0.5$ kg/m^3 s, $Q_m = 420$ W/m^3, $T_f = 100$ °C, $V = 300$ cm^3/s, $\varphi = 0.3$, $T^* = 3$ s).

water in the tissue may affect the thermal conductivity of the respiratory tract. Usually, the thermal conductivity of tissue will decrease with the loss of water. This may imply that the thermal injury will be more serious if taking into concern the decrease of the thermal conductivity due to water loss. Fig. 9 gives the temperature responses in tissue at a specific position ($z = 8$ cm, $r = 0.635$ cm) with different metabolic rates. Clearly, the larger the metabolic rate, the higher the temperature increases. This suggests that keeping calm is helpful to minimize the thermal injury in respiratory tract. It is noticed that the blood perfusion rate, W_b, is correlated with metabolic heat generation rate Q_m, which can be approximately expressed as [20]:

$$W_b = Q_m \times 10^{-3}\ \mathrm{kg/J} \tag{6}$$

Fig. 7 indicates that large blood perfusion tends to prevent the tissues from burn injury. Thus, the tissue temperature

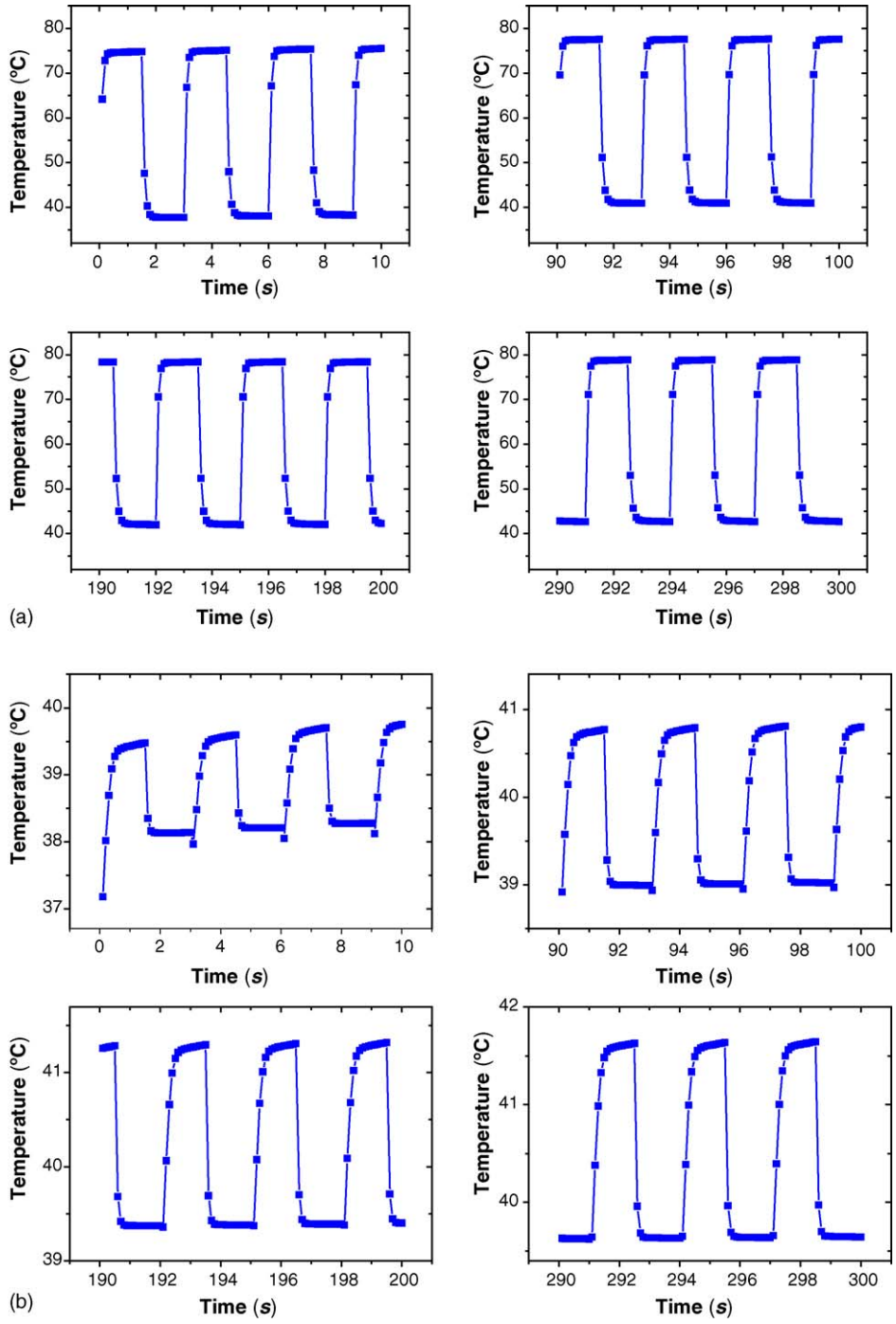

Fig. 6. Transient air temperature responses at two sections $z = 8$ cm (a) and z = 52 cm (b) at four time segments ($K = 0.5$ W/m °C, $W_b = 0.5$ kg/m^3 s, $Q_m = 420$ W/m^3, $T_f = 100$ °C, $V = 300$ cm^3/s, $\varphi = 0.3$, $T^* = 3$ s).

will be in fact a little smaller than that shown in Fig. 9 at the same metabolic rate if we consider the blood flow increase with the metabolism. However, the difference will not be substantial because the blood flow in the surface of respiratory tract is very low and does not change very much during increased metabolism.

In order to supply enough oxygen for respiration, the respiratory rate tends to increase after exposure in a fire. As shown in Fig. 10, the tissue mean temperatures do not display evident differences under various respiratory rates. It is noticed that the amplitude of the temperature oscillation can increase with decreases in the respiratory rate. With the decrease of the respiratory cycle (RC), heat received by the respiratory air from the deeper tissue of nasal mucosal is not sufficiently large because of the high-speed flowing air during the inspiratory and expiratory phases of the breathing cycle. Further calculation indicates that relative humidity of the surrounding air has no significant effect on the thermal

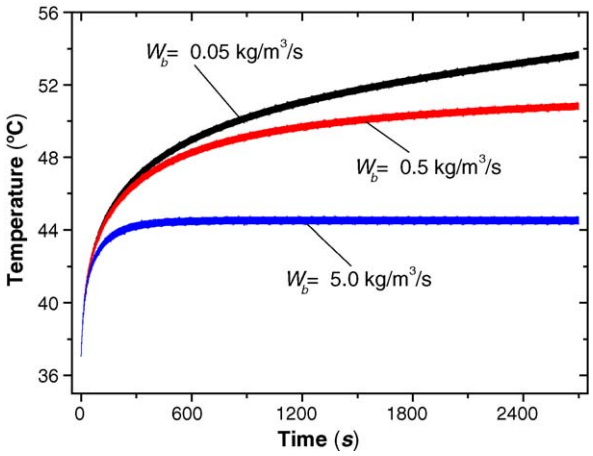

Fig. 7. Effect of blood perfusion levels on temperature response at a specific position ($z = 8$ cm, $r = 0.635$ cm) ($K = 0.5$ W/m °C, $Q_m = 420$ W/m^3, $T_f = 100$ °C, $V = 300$ cm^3/s, $\varphi = 0.3$, $T^* = 3$ s).

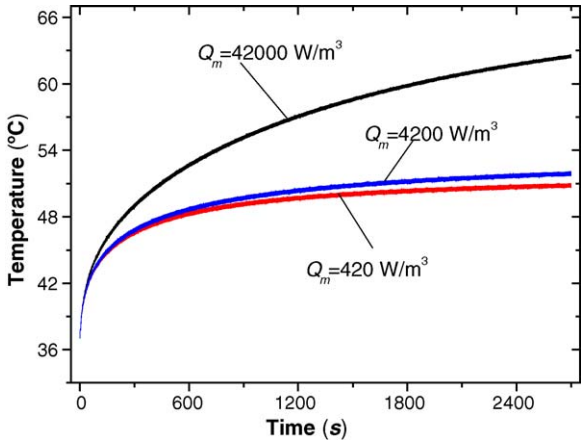

Fig. 9. Effect of metabolic rate of tissues on temperature response at a specific position ($z = 8$ cm, $r = 0.635$ cm) ($K = 0.5$ W/m °C, $W_b = 0.5$ kg/m^3 s, $T_f = 100$ °C, $V = 300$ cm^3/s, $\varphi = 0.3$, $T^* = 3$ s).

injury in the respiratory tract for short duration exposures. The reason lies in that the heat loss at the mucosal surface due to evaporation is much less than that due to convection when the surrounding air is dry and hot.

Apart from the above factors, the environmental variables, such as the surrounding air temperature also plays a key role in deciding the thermal injury. Extremely hot environments will cause quick thermal injury in the respiratory tract. For the case of $T_f = 150$ °C, the temperature of tissue at $z = 8$ cm, $r = 0.635$ cm will become 53.9 °C at 300 s. However, when the surrounding air temperature is 70 °C, the temperature of tissue at the same position is only 41.9 °C (Fig. 11). As it appears, the temperature of the surrounding air has an important effect on the thermal injury in respiratory tract. A healthy man will lose consciousness quickly when the surrounding air temperature reaches about 150 °C [21].

Fig. 12 illustrates the effect of local mean longitudinal air velocity on temperature response at a specific position ($z = 8$ cm, $r = 0.635$ cm). Different from the effect of the respiratory rate, a higher air velocity can increase the tissue temperature absurdly. From this point, decreasing the air velocity and increasing respiratory rate is helpful for minimizing the thermal injury in respiratory tract. For the same reason, increasing the cooling air velocity and decreasing respiratory rate may help alleviate the burn injury of human respiratory tract due to inhalation of hot gas at the early stage of fires.

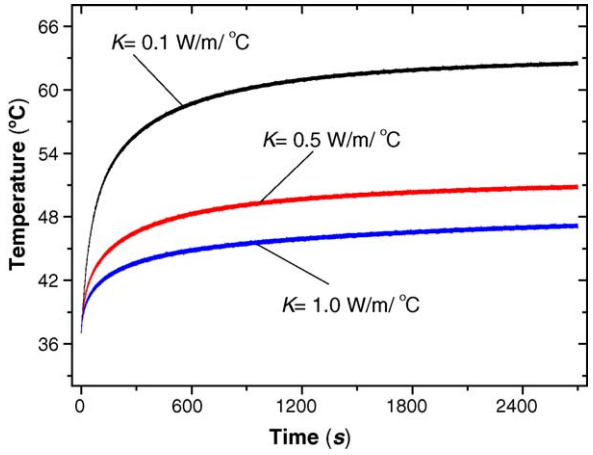

Fig. 8. Effect of thermal conductivity of tissues on temperature response at a specific position ($z = 8$ cm, $r = 0.635$ cm) ($W_b = 0.5$ kg/m^3 s, $Q_m = 420$ W/m^3, $T_f = 100$ °C, $V = 300$ cm^3/s, $\varphi = 0.3$, $T^* = 3$ s).

3.4. Burn evaluation

It is generally accepted that thermal damage begins when the tissue temperature rises above 44 °C [22]. Quantitative burn degree evaluation was first proposed by Henriques and Moritz [23] based on that the tissue damage can be represented as an integral of a chemical rate process

$$\Omega = \int_0^t P \exp\left(-\frac{\Delta E}{RT}\right) \mathrm{d}t \tag{7}$$

where P is a constant that varies with tissue and local temperature, ΔE and R are the activation energy and idea gas constant, as given in Table 1. If both conditions of $T > 44$ °C and $\Omega > 0.53$ are satisfied at the entrance of the human respiratory tract ($z = 0$), then it is defined as the first-degree burn. In the following, burn times are predicted based on the integral constants given by Henriques and and Moritz [23]. Fig. 13 is the dimensionless Henriques' burn integral distribution at the surface of tissue ($0.635\ \text{cm} \leq r \leq 3.635$ cm) during a fire. As shown in Fig. 13, Ω at the inlet of the nasal cavity increases from 0 to 40 within 800 s. Obviously, due to the surface water evaporation cooling, the burn injury often occurs at certain positions underneath the skin surface near the inlet of respiratory tract. Most of the tissues near the surface suffer injury immediately after the exposure, while in the deeper tissues, serious damage occurs after a relatively longer time period. Fig. 14

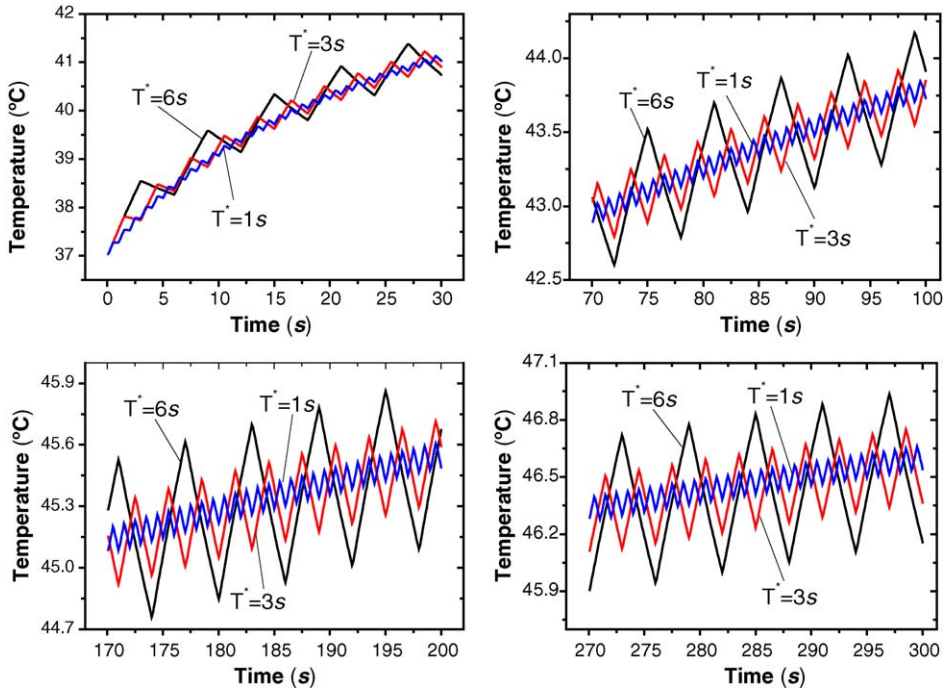

Fig. 10. Effect of respiratory frequency on temperature response at a specific position ($z = 8$ cm, $r = 0.635$ cm) ($K = 0.5$ W/m °C, $W_b = 0.5$ kg/m^3 s, $Q_m = 420$ W/m^3, $T_f = 100$ °C, $V = 300$ cm^3/s, $\varphi = 0.3$).

indicates the first-degree burn time predicted by Eq. (7). At the nasal mucosal surface ($r = 0.635$ cm), the first-degree burn will occur only after 230 s, while more than 900 s is needed for the first-degree burn to occur at a deeper tissue ($r = 0.935$ cm). All these differences are caused by the thermal development of the hot air heating. Clearly, facing the fire, the human upper respiratory tract will be easily subject to serious burn if getting away from the fire ground was not successful. With the help of the theoretical predicted results, clinicians could quickly evaluate the burn degree after they get to know the time that patient subject to a fire

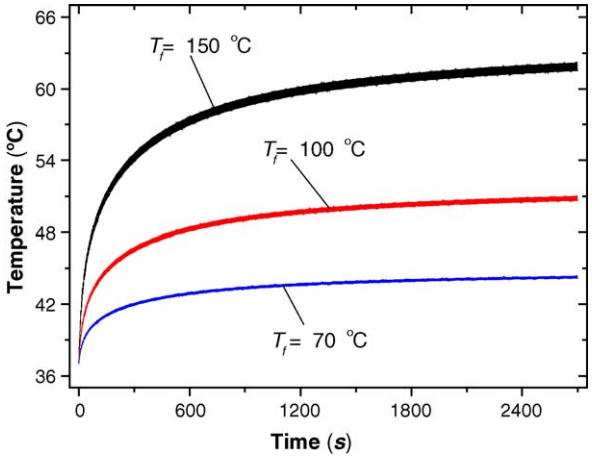

Fig. 11. Effect of surrounding air temperature on temperature response at a specific position ($z = 8$ cm, $r = 0.635$ cm) ($K = 0.5$ W/m °C, $W_b = 0.5$ kg/m^3 s, $Q_m = 420$ W/m^3, $V = 300$ cm^3/s, $\varphi = 0.3$, $T^* = 3$ s).

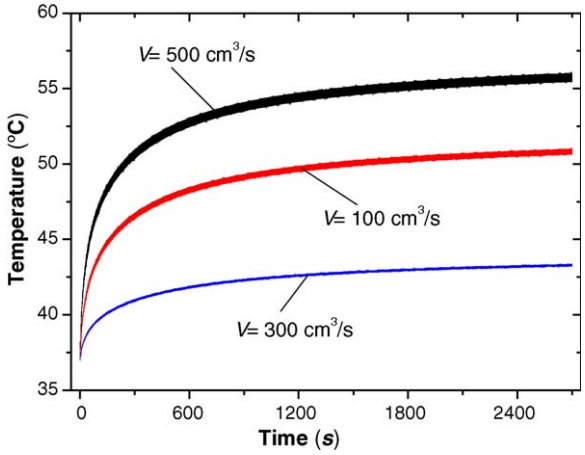

Fig. 12. Effect of local mean longitudinal air velocity on temperature response at a specific position ($z = 8$ cm, $r = 0.635$ cm) ($K = 0.5$ W/m °C, $W_b = 0.5$ kg/m^3 s, $Q_m = 420$ W/m^3, $T_f = 100$ °C, $\varphi = 0.3$, $T^* = 3$ s).

Table 1
Constant for the first- and second-degree burns integral [1]

Epidermis	Henriques' constant	Weaver's constant	Mehta's constant
P (s^{-1})	3.1×10^{98}	3.1×10^{98}, 44 °C $< T \leq$ 50 °C 1.823×10^{51}, $T \geq 50$ °C	1.43×10^{72}
$\Delta E/R$ (K)	75000	93534.9, 44 °C $< T \leq$ 50 °C 39109.8, $T \geq 50$ °C	55000

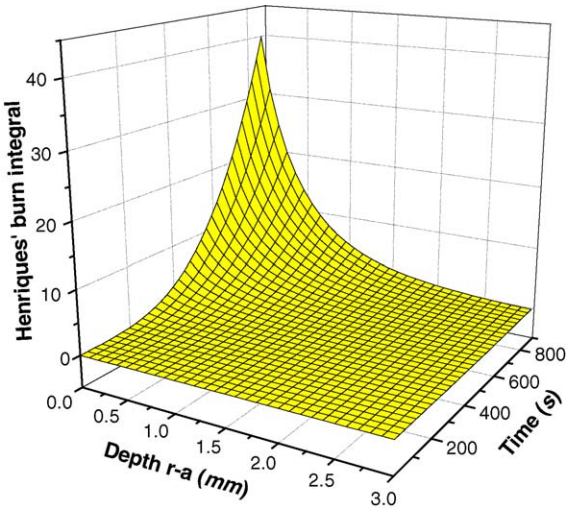

Fig. 13. Dimensionless Henriques' burn integral distribution at the surface of tissue (0.635 cm $\leq r \leq$ 3.635 cm) during a fire ($z = 0$, $K = 0.5$ W/m °C, $W_b = 0.5$ kg/m^3 s, $Q_m = 420$ W/m^3, $T_f = 100$ °C, $V = 300$ cm^3/s, $\varphi = 0.3$, $T^* = 3$ s).

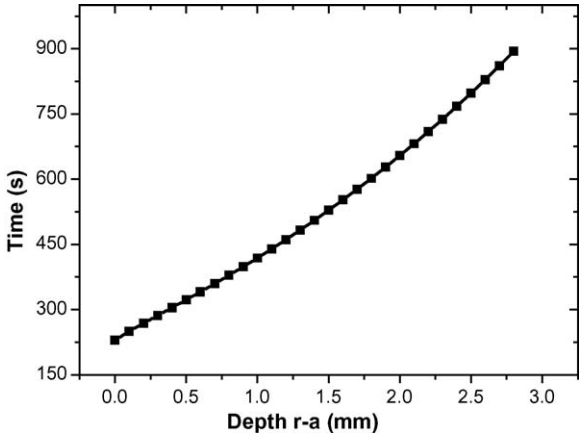

Fig. 14. Time prediction of the first-degree burn at the surface of tissue (0.635 cm $\leq r \leq$ 3.635 cm) during a fire ($z = 0$, $K = 0.5$ W/m °C, $W_b = 0.5$ kg/m^3 s, $Q_m = 420$ W/m^3, $T_f = 100$ °C, $V = 300$ cm^3/s, $\varphi = 0.3$, $T^* = 3$ s).

and the approximate temperature of a fire. This might be beneficial for the doctor to adopt an appropriate treatment protocol.

4. Conclusion

In order to predict the air temperature response in the nasal cavity, a transient theoretical model was described to interpret the thermal injury at the early stage of fires. Effects of the individual's physiological and environmental variables (such as blood perfusion, thermal conductivity, metabolic rate, air temperature, air velocity, and respiratory rate) were investigated in detail. According to the numerical results, decreasing the air velocity and increasing respiratory rate is helpful to minimize the thermal injury in respiratory tract. The effect of relative humidity of surrounding air can be ignored in predicting burns for short duration exposures. Further, burn times are predicted based on the classical evaluation criterion. Most of the tissues near the surface suffer injury immediately after the exposure, while in the deeper tissues, serious damage occurs after a relatively longer time period. However, several limitations in this study should be pointed out. For example, more complex geometrical and physiological parameters of the respiratory system may be incorporated for a more comprehensive prediction. The present model, mainly developed here for characterizing the situations at early stage of fire, still could not be directly used to study the heat transfer process when the nasal tract has been completely burned. In that case, tissue parameter and the equations need to be modified. Further, the present model had not included the effects of the densely wet hot air such as the inhaled water vapor. For that purpose, phase change heat transfer must be considered to address such water evaporation behavior. These problems will be addressed in our later research. Studies performed in this paper warrant further investigation along this direction.

Acknowledgement

This work is partially supported by the National Natural Science Foundation of China under grant 50325622.

References

[1] Pruitt Jr BA, Goodwin CW, Mason Jr AD. Epidemiological, demographic and outcome characteristics of burn injury. In: Herndon DN, editor. Total burn care. London/Edinburgh/New York/Philadelphia/St. Louis/Sidney/Toronto; 2002. p. 16–32.
[2] Sarhadi NS, Murray GD, Reid WH. Trends in burn admissions in Scotland during 1970–1992. Burns 1995;21:612–5.
[3] Carvajal HF. Fluid therapy for the acutely burned child. Comp Ther 1977;3:17–24.
[4] Curreri PW, Richmond D, Marvin J, Baxter CR. Dietary requirements of patients with major burns. J Am Diet Assoc 1974;65: 415–7.
[5] Hildreth MA, Herndon DN, Desai MH, Duke MA. Reassessing caloric requirements in pediatric burn patients. J Burn Care Rehabil 1988;9:616–8.
[6] Jackson MP, Philp B, Murdoch LJ, Powell BWEM. High frequency oscillatory ventilation successfully used to treat a sever paediatric inhalation injury. Burns 2002;28:509–11.
[7] Herndon DN, Barrow RE, Rutan RL, Rutan TC, Desai MH, Abston S. A comparison of conservative versus early excision. Therapies in severely burned patients. Ann Surg 1989;209:547–53.
[8] Wilmore DW, Long JM, Mason Jr AD, Skreen RW, Pruitt Jr BA. Catecholamines: mediator of the hypermetabolic response to thermal injury. Ann Surg 1974;180:653–69.
[9] Sheridan R. Specific therapies for inhalation injury. Crit Care Med 2002;30:718–9.
[10] Guy JS, Peck MD. Smoke inhalation injury: pulmonary implications. Respir Care 1999;3:904–12.
[11] Demling RH. Smoke inhalation injury. In: Shoemaker W, editor. Textbook of critical care. PA, USA: WB Saunders Company; 1995 p. 1506.

[12] Moritz AR, Hendriques FC, McLean R. The effects of inhaled heat on the air passages and lungs-an experimental investigation. M J Path 1945;2:311–31.
[13] Moritz AR, Weisinger JR. The effects of cold air on the sie passages and lungs. Arch Intern Med 1945;75:233–40.
[14] Hanna LM, Scherer PW. A theoretical model of localized heat and water vapor transport in the human respiratory tract. ASME J Biomech Eng 1986;108:19–27.
[15] Hanna LM, Scherer PW. Measurement of local mass transfer coefficients in a cast model of the human upper respiratory tract. ASME J Biomech Eng 1986;108:12–8.
[16] Nuckois ML, Zumrick JL, Johnson CE. Heat and water vapor transport in the human upper airways at hyperbaric conditions. ASME J Biomech Eng 1983;105:24–30.
[17] Saidel GM, Kruse KL, Primiano FP. Model simulation of heat and water vapor transport dynamics in an airway. ASME J Biomech Eng 1983;105:188–93.
[18] Liu J, Wang C. Bioheat transfer, 1st ed., Beijing: Science Press; 1997 [in Chinese].
[19] Zhao RY, Fan CY, Xie DH, Qian YM. Air conditioning, 3rd ed., Beijing: Architecture and Building Press; 1998 [in Chinese].
[20] Wang C. Relationship between the basal metabolic rate and temperature regulation of human being and animals. In: Proceedings of international symposium on macro- & micro-scopic heat and mass transfer in biomedical engineering; 1991.
[21] Li A, Yang ZY. Inhalation injury, 1st ed., Beijing: People's Military Medical Press; 1993 [in Chinese].
[22] Torvi DA, Dale JD. A finite element model of skin subjected to a flash fire. ASME J Biomech Eng 1994;116:250–5.
[23] Henriques FC, Moritz AR. Studies of thermal injury. I. The conduction of heat to and through skin and the temperatures attained therein. A theoretical and an experimental investigation. Am J Pathol 1947;23: 531–49.